

REACTION TIME AND PUPILLARY DILATION MEASURES OF EMOTIONAL
INFORMATION PROCESSING IN DYSPHORIA

BY

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Abstract

Subclinical depressive syndromes such as dysphoria represent increased risk for developing depression and can lead to deleterious mental and physical health outcomes similar to those associated with major depression. Thus, investigating relationships between cognitive processing and dysphoria is important. Studies utilizing sustained pupil dilation as a psychophysiological index of information processing have suggested that depressed individuals process emotional and perhaps specifically negative information for longer than nondepressed individuals. In the current study, 29 dysphoric and 33 nondysphoric individuals completed an emotional information processing task, and pupil dilation and reaction time (RT) data were compared to investigate whether depressotypic phenomena might be detectable prior to the development of depression. The hypothesized dysphoria status by stimulus valence interaction was unsupported. Unexpectedly, gender interacted with dysphoria status to account for variance in pupil dilation and RT. In males, dysphoria had a facilitative effect on early processing of emotional information and appeared to reduce late processing associated with positive stimuli. However in women, dysphoria briefly interfered with the typical female advantage for emotional information processing and increased sustained processing following negative stimuli. Thus, increased risk for depression may be expressed via non-identical cognitive mechanisms in dysphoric men and women. Results suggest that behavioral and physiological investigations intending to elucidate relationships between

emotional information processing and vulnerability to depression should not fail to explore gender as a potentially important interacting variable.

Reaction time and pupillary dilation measures of emotional information processing in dysphoria

Major depression is among the most prevalent psychological disorders, and it leads to a well-documented variety of deleterious mental and physical health outcomes. Lifetime risk estimates for Major Depressive Disorder have been reported to be 25% for women and 12% for men (American Psychiatric Association, 2000). Evidence from epidemiological studies suggests that the prevalence of depression is increasing (De Marco, 2000), and that the average age of onset—currently the mid-20s—is decreasing in recent years (American Psychiatric Association, 2000). Globally, depression is among the leading causes of disability and suicide (World Health Organization, 2006).

Biological studies of depression have revealed that genetics moderately contribute to depression and depression risk (Kendler, Kessler, Neale, Heath, & Eaves, 1993; Kendler, Neale, Kessler, Heath, & Eaves, 1992; McGuffin, Katz, & Rutherford, 1991), and in turn, depression is usually accompanied by observable biological sequelae. A current depressive episode is often associated with disturbances of normal sleep, appetite, and cognitive functioning. In addition, long-term and recurrent depression have been linked to neuron loss in the hippocampus (Sapolsky, 2000), a brain structure critical to memory. Depression is also related to diminished immune system response (Thase, Jindal, & Howland, 2002).

Psychosocially, depressed individuals experience frequent rejection (Segrin & Abramson, 1994) and have lower quality and less satisfying relationships (Segrin,

2001). In terms of functional correlates, depression is associated with subsequent unemployment and loss of family income at the individual level (Whooley et al., 2002), and high healthcare and loss-of-productivity costs on the societal level. In the U.S. alone, yearly costs associated with depression have been calculated to be in excess of \$80 billion (Greenberg et al., 2003), an estimated \$23 billion of which is due to depressive symptoms that do not meet the full diagnostic criteria for MDD.

Current estimates suggest that 60% of individuals who have one depressive episode will have a second. As the number of episodes experienced increases to two or three, the chance of having a subsequent episode increases to 70% and 90% respectively (American Psychiatric Association, 2000; Solomon et al., 2000; Stewart, Ricci, Chee, Hahn, & Morganstein, 2003). Thus, the prevention of first onsets of major depression is of critical concern on the individual and societal levels, and underscores the importance of identifying factors that may increase the risk of developing depression.

Cognitive Theories of Depression

Cognitive theories of depression have provided valuable perspectives from which to investigate the potential causes and correlates of depression. Early cognitive theories proposed that maladaptive thinking, such as biased negative interpretations and irrational beliefs, contribute to the onset of depression (Beck, 1967; Ellis, 1962). In particular, Beck (1967; Beck, Rush, Shaw, & Emery, 1979) theorized that the activation of negative cognitive schemas causes the individual to filter and construe his or her experiences in a negative manner. This ultimately leads to what Beck calls

“the cognitive triad,” a pattern of depressive thinking consisting of negative thoughts about the self, the world, and the future. Other influential cognitive perspectives have implicated learned helplessness and attribution processes (Abramson, Seligman, & Teasdale, 1978; Seligman, 1978), and cognitive structures such as negative self-concept, as factors causally related to depression (Beck, 1967; Blatt, 1974).

Information Processing in Depression

Cognitive conceptions of depression have come to incorporate the information processing approach, which seeks to understand human behavior in terms of how information is gathered, modified, encoded, and used (Ingram, 1984; Merluzzi, Rudy, & Glass, 1981; Neisser, 1967). Through such techniques, differential biases in selective attention and memory have been documented in depressed individuals compared to nondepressed individuals. It has long been proposed that an information processing analysis of depression consists of key theoretical cognitive constructs, including network theories, affective structures, depth of processing, and cognitive processing capacity (e.g., Ingram, 1984).

Network theories have suggested that memories consist of cognitive networks of associated concepts, (Collins & Loftus, 1975) and that clusters of similarly related concepts, or “nodes” (Bower, 1981) are activated by either environmental stimuli or through the spread of activation from other associative connections. Bower (1981) further proposed the existence of theoretical affective structures, or “nodes”, which consist of associated memories, concepts, and experiential aspects of an emotion (i.e., depression), and are presumably connected to any number of other thoughts and

memories, such as situations or experiences that can evoke the emotion. In addition, it is thought that the depth to which information is processed dictates the degree to which an individual perceives, processes and encodes information into memory. It has also been proposed that a greater proportion of cognitive processing capacity—the finite amount of attention that can be paid to selective elements from an array of internal or external stimuli—is needed for deep and elaborated processing. Therefore, the more intricate one’s network of depressive associations and the more likely one is to think deeply or persistently about sad thoughts and memories (i.e., rumination), the greater the proportion of one’s cognitive processing capacity will be usurped. From this perspective, major depression could be characterized by frequent activation of depressive nodes, which leads to thinking about additional sad associations, which then propagates further elaboration of sad thoughts.

The Interacting Cognitive Subsystems (ICS) framework has since modified earlier information processing analyses of depression (Teasdale, 1997; Teasdale & Barnard, 1993). The ICS framework suggests that lower-order specific *propositional* representations and higher-order, general *implicational* representations (i.e., schematic models) of information form an interlocking feedback processing loop in depression. For instance, an individual might encounter specific propositional information (e.g. “I failed the test”), which contributes to a general implicational representation (e.g. “I am a total failure”) that may have already been predisposed by negative mood, negative events earlier that day, or feelings of physical sluggishness.

In turn, that same individual's implicational schematic model may output specific meanings forecasting the high probability of failure in upcoming endeavors.

Reciprocal cycles of propositional-implicational processing are involved in a variety of controlled processing tasks, and have been termed the “central engine” of cognition (Teasdale & Barnard, 1993, pp. 76-81). The processes that transform schematic models into propositional meanings, and vice versa, are vulnerable to processing capacity limitations. This means that when multiple information streams compete for use of the same processing resources, one is selected, and other streams cannot be processed.

The ICS analysis also suggests that emotional responses can only result from implicational level processing, which experientially resembles “intuition” and “feeling” more than logically specific premises. Of some relevance, LeDoux (1996) has proposed a functional neuroanatomical model in which imbalanced parallel processing of emotional aspects (via the amygdala system) and nonemotional aspects (via the hippocampus) of information could combine to generate dysfunctional behavior. Although these models were not necessarily intended to be complementary, each implies that different mechanisms may be involved in processing emotional information and nonemotional information. Furthermore, the models suggest that deeply meaningful, persistent depressive cognition can lead to cognitive deficits (i.e., interference) in performing tasks that demand greater effortful processing.

Based on the aforementioned information processing theories, investigators have designed experiments that operationalize, measure, and compare selective

attention and memory processing of depressed and nondepressed individuals. Studies in which dysphoric and depressed individuals selectively attend to and remember negative information (Blaney, 1986; Matt, Vazquez, & Campbell, 1992; Matthews & Harley, 1996; J. M. Williams, Mathews, & MacLeod, 1996) support the notion that these processing biases are characteristic phenomena of dysphoria and depression. Depressed and dysphoric individuals have also been found to identify negatively valenced words significantly faster than positively valenced words, unlike nondepressed individuals (Siegle, et al., 2001; Siegle, Ingram, et al., 2002). This could reflect greater accessibility to negative information while in a congruently negative mood (i.e., activated depressive node and associated memories), or it might reflect greater efficiency of neural structural processing that has developed through repetitive use of synaptic pathways over time (i.e., Hebb learning).

Several cognitive theories have specifically implicated biased attention to *emotional* information as being essential to the onset and maintenance of depression (Beck, 1967, 1974; Ingram, 1984; Ingram, Miranda, & Segal, 1998). In order to test this idea empirically in depressed and dysphoric individuals, investigators have compared the attentional processing of emotional versus semantic aspects of information (Siegle, Granholm, Ingram, & Matt, 2001; Siegle, Ingram, & Matt, 2002). A single word such as “award” can be conceived as having a nonemotional semantic meaning as well as a positive emotional meaning. Theories of emotional information processing posit that the emotional and nonemotional aspects of information are processed in parallel through highly interconnected physiological and

cognitive systems (Bower, 1981; Ingram, 1984; LeDoux, 1996; Siegle, et al., 2002) Attention can be differentially focused on either aspect of the information (Kitayama, 1990; Matthews & Harley, 1996). If it is accepted that emotional and nonemotional aspects of information are processed in parallel, the degree to which attention is disproportionately allocated to emotional information may correspondingly hinder the processing of semantic information.

Siegle and colleagues (2002) observed this phenomenon in dysphoric individuals and termed it “affective interference.” Carrying this a step further in the context of an example, we can conceive of a person who disproportionately focuses on the negative emotional aspects of a word (e.g., “decaying”), which triggers other negative associations that are unrelated to the semantic meaning of the word (e.g., “shame”). Thus, activation of depressive associations could spread via negatively connoted words. In this way, exposure to negatively connoted stimuli could lead to a persistent pattern of elevated negative thinking, and perhaps increased susceptibility to depressive states.

Cognitive Vulnerability to Depression

Theories of cognitive vulnerability suggest that characteristic patterns of cognitive processing contribute to the development, maintenance, and recurrence of major depressive episodes. Research attempting to identify cognitive vulnerability factors for depression is embedded in a diathesis-stress conceptualization. Although it is widely accepted that biases in selective attention and memory correlate with current depressive episodes, cross-sectional data do not permit the inference that these biases

contribute to the development of depression. Furthermore, research has shown that most depressive processing biases dissipate with the remission of depressive symptoms. Without further evidence to the contrary, it would be plausible to view these biases as consequences of depression rather than contributing vulnerability to depression. However, diathesis-stress models of depression theorize that depressogenic cognitive schemas remain inactive until they are triggered by stressful events (Beck, 1967; Ingram, 1984; Ingram, et al., 1998; Teasdale, 1983). Thus, a nondepressed individual may possess latent depressive cognitive schemas that only become activated under sufficient distress, which would predispose him or her to developing a more stable depressive episode. Studies utilizing mood-priming designs have largely supported the idea that formerly but not currently depressed individuals exhibit evidence of depressive schemas and attentional biases for negative information (Ingram, Bernet, & McLaughlin, 1994), which are activated by sad mood, and which may make them more vulnerable to developing a future depressive episode (Scher, Ingram, & Segal, 2005).

Mood-priming studies of formerly depressed individuals provide an important though limited perspective on cognitive vulnerability. These designs cannot rule out the possibility that any observed schema-activated cognitive correlate is actually a consequence, or “scar”, of a past depressive episode. From this perspective, a correlate like depressive selective attention could logically occur during or following an episode, but not prior to a first onset of depression. To circumvent this issue, vulnerability research may look to prospective studies to determine which factors

precede the first onset of a disorder. However, the substantial cost and time required for these studies makes them somewhat prohibitive and less common. Nonetheless, prospective studies such as the Temple-Wisconsin Cognitive Vulnerability to Depression Project have contributed important evidence that specific cognitive vulnerability factors exist prior to first onsets of depression (Alloy et al., 2004).

An alternative cross-sectional approach is to measure a known correlate of depression and establish its occurrence in a high-risk sample, (e.g., subclinical depression or dysphoria), prior to a first onset of major depression. Of course, without longitudinal follow-up there is no way to determine how many of these high-risk individuals subsequently develop depression. Nonetheless, given the diathesis-stress model assumptions noted earlier, a depressed mood state could potentially activate cognitive vulnerability factors, which in turn, could maintain or exacerbate the depressed mood and other depressive symptoms. With increased number and severity of symptoms the threshold of major depression could be crossed. It should be noted that this approach implicitly assumes that nosologically differentiated major depressive episodes and subclinical depressive syndromes are not discrete categorical entities, but that they are more dimensionally related.

There is growing acknowledgement that depressive symptoms may fall along a continuum (Akiskal, Judd, Gillin, & Lemmi, 1997; Angst & Merikangas, 1997; Angst, Merikangas, & Preisig, 1997; Hudson & Pope, 1990; Lewinsohn, Solomon, Seeley, & Zeiss, 2000), and that subclinical depressive syndromes (i.e., a period in which some but not sufficient DSM-IV-TR diagnostic criteria for major depressive

episode are met) can be associated with similar deleterious outcomes as major depression. For example, subthreshold depressive symptoms can be associated with significant psychosocial dysfunction, functional impairment, and increased risk for substance abuse (Gotlib, Lewinsohn, & Seeley, 1995). Also, the experience of subclinical depressive symptoms appears to significantly increase risk for major depression (Cuijpers & Smit, 2004).

Evidence indicates that subclinical depressive symptoms can strongly predict an episode of major depression in the long-term (Angst & Merikangas, 1997; Cuijpers & Smit, 2004; Lewinsohn et al., 2000; Pine, Cohen, Cohen, & Brook, 1999). It has even been suggested that all individuals who develop MDD pass through a period characterized by a subclinical depressive syndrome (Cuijpers & Smit, 2004). Prospective studies have found that individuals with current subthreshold depression have up to 6-times the relative risk of developing MDD than individuals who have never experienced subthreshold episodes (Eaton, Badawi, & Melton, 1995; Warner, Weissman, Fendrich, Wickramaratne, & Moreau, 1992). Furthermore, recent studies have shown that treating individuals with subclinical depressive symptoms can reduce the number of new cases of MDD (Clarke et al., 1995; Clarke et al., 2001).

Dysphoria is similar to—but should not be considered equivalent to—subthreshold depression. It is a generalized negative affective syndrome that characterizes almost any depressive state (Ingram & Hamilton, 1999) and is a central feature of depressive disorders. However, dysphoria can consist of depressive symptoms (e.g., sad mood, anhedonia) *and* symptoms not exclusive to depression

(e.g., anxiety). Dysphoria tends to be less severe, less enduring, and, as indicated, less affectively specific than depressive disorders as they are diagnosed by classification systems (Ingram & Wisnicki, 1999). Nonetheless, dysphoria may be a vulnerability factor for developing MDD or other more severe disorders (Haaga & Solomon, 1993).

The logical assumption that dysphoria is far more common than diagnosable depressive disorders (Ingram & Hamilton, 1999) underscores the importance of learning about characteristics and correlates of this syndrome. Haaga and Solomon (1993) have asserted, and Ingram and Hamilton (1999) have reiterated, that individuals with subclinical depressive features comprise an ideal population if risk processes are the focus of investigation. Along similar lines, Persons (1986) argued that searching for pathological mechanisms of symptoms or syndromes as they occur naturally has advantages over searching for mechanisms underlying diagnostically categorized disorders. In particular, a focus on symptoms or syndromes enables investigators to isolate components of psychopathology in ways that might be ignored by diagnostic classification systems, to examine the continuity of psychological symptoms with nonpathological phenomena, to refine the development of diagnostic classification systems, and to inform psychological theory. Persons (1986) points to *learned helplessness theory of depression* as an example of a theory derived from an observed group of motivational, cognitive, and emotional behaviors (Seligman, 1978) rather than diagnostic criteria of depression. Accordingly, the study of dysphoria

provides greater flexibility in examining a naturally occurring, theoretically continuous phenomenon, which also has the potential to inform depression theories.

Dysphoria is most often measured through the *Beck Depression Inventory* (BDI). It has been proposed that scores in the range of 10-17 indicate the presence of dysphoria. This operationalization was originally suggested to identify those individuals whose composite symptom severity score on the BDI was below the criterion for major depression but higher than the normative range (Kendall, Hollon, Beck, Hammen, & Ingram, 1987).

Although BDI scores reflect a dimensional approach to measuring severity (as opposed to a categorical approach), there is a lack of clear evidence that incremental increases in BDI scores correlate systematically with other known markers of dysphoria or depression (Kendall, et al., 1987). Thus, presuming the BDI to be a highly sensitive analogue to “continuous” dysphoria severity would be inaccurate.

Haaga and Solomon (1993) have also identified problems with deriving hypotheses from depression theory by using solely the BDI to measure dysphoria, without also assessing purer markers of negative affectivity. As noted earlier, dysphoria can include symptoms of depression *and* anxiety. Major depression is characterized by both increased negative affect and decreased positive affect, whereas anxiety is associated with increased negative affect, but not specifically with decreased positive affect (Clark & Watson, 1991). Thus, if an individual’s dysphoria is characterized by subclinical depressive features, decreased positive affect might be observed in addition to increased negative affect. Measures such as the *Positive and*

Negative Affect Schedule (PANAS), and the *Beck Anxiety Inventory* (BAI) can be used to isolate positive affect from negative affect, and discriminate anxiety from depression, respectively. While the high comorbidity of symptoms of depression and anxiety in community settings makes the search for “purely depressive” research participants somewhat impractical, a multi-measure approach might facilitate a more sophisticated post hoc analysis of dysphoria in a given study.

Dysphoria is often referred to as a state, but it is thought to have a higher degree of stability than a passing mood. On the other hand, dysphoria is thought to be far less enduring than major depression, which if left untreated lasts an average of 5-6 months (Lehmann, 1983). Nonetheless, the fact that dysphoric individuals have been found to disproportionately attend to (Siegle, Ingram, et al., 2002) and remember (Matt, et al., 1992) negative emotional information (i.e., similar to depressed individuals) suggests that dysphoria is more than a transient mood state. As such, behavioral and physiological techniques developed to assess cognitive and emotional processing have become important tools for studying both depression and dysphoria.

Pupil Dilation and Cognitive and Emotional Processing

The direct measurement of behavioral and physiological activity tied to information processing tasks enables investigators to measure, more precisely and in real-time, operationalizations of information processing. One relatively novel method, pupillometry, assesses sustained pupil dilation in response to the presentation of particular stimuli. This technique involves measuring the diameter of the pupil over time. Although earlier methods facilitated the measurement of participants’ stimulus-

to-response reaction times for between-group comparisons, pupillometry has introduced an important temporal processing dimension. Pupil dilation has become an accepted index of cognitive (Beatty, 1982; Steinhauer & Hakerem, 1992) and emotional processing (Janisse, 1974; Szabadi & Bradshaw, 1996). In an early review of the pupil dilation literature, Janisse (1974) confirmed that pupil dilation increases with the intensity of stimulation, regardless of whether it is positive or negatively valenced. In another review of experimental data on task-evoked pupillary response, Beatty (1982) concluded that pupillary response fulfills the criteria of a physiological measure of processing load, or “mental effort,” in that it accurately reflects within-task, between-task, and between-individual variations in processing demands. Further, Granholm, Asarnow, Sarkin, and Dykes (1996) found that pupillary dilation increased systematically with increasing processing demands up to resource limits during a digit span recall test (i.e., having to hold in memory each additional digit). Steinhauer and Hakerem (1992) discussed the pupillary response’s relation to neurological signs of cognitive activity such as event-related potentials, and they suggested that pupil dilation resulting from information delivery provides “useful adjuncts in the study of psychopathology” (p. 182). Although much of the work they reviewed centers on abnormal pupillary dilation responses related to schizophrenia and schizophrenia-vulnerability, they suggested that studying the pupil allows access to cognitive mechanisms, and could potentially identify vulnerability to other forms of psychopathology.

Sustained Pupil Dilation and Depression

Siegle and colleagues have since demonstrated that the assessment of pupillary dilation is a promising approach for studying information processing in individuals with depression. In particular, studies have utilized an emotional valence identification (VID) task in which depressed and never depressed participants classify words presented on a computer screen as positive, negative or neutral, as quickly as possible. While performing this task that focuses on emotional information, depressed individuals have exhibited greater sustained pupil dilation than never depressed individuals (Siegle et al., 2001; Siegle, Steinhauer, Carter, Ramel, & Thase, 2003). This sustained dilation is observed 4-5 seconds after the presentation of an emotional stimulus (Siegle, et al., 2001). Sustained pupil dilation can be thought to reflect deeper processing and more broadly diffused neural activation. Findings from these studies suggest that depressed individuals process emotional, negative and personally relevant information for a longer duration than nondepressed individuals do. Sustained elaborative cognitive processing (e.g., generating associations) is presumed to play an important role in the cognitive biases exhibited by individuals with depression (MacLeod & Mathews, 1991; J. M. G. Williams & Oaksford, 1992).

Certain self-report measures of rumination have also been found to correlate moderately with sustained pupil dilation to negative personally relevant emotional information (Siegle et al., 2003). These findings are especially significant considering the fact that depressed individuals appear to show significantly *less* sustained pupil dilation than never-depressed individuals on tasks that focus on nonemotional stimuli such as the Stroop color-word task (Siegle, et al, 2004). It is possible that the

increased elaborative processing of emotional information and decreased elaborative processing of nonemotional information underlies the kinds of real world functional impairment people experience, such as reduced task-relevant focus and job productivity.

It is also possible that sustained processing may represent a precursor to or early building blocks of rumination (Siegle, et al., 2003). Evidence has suggested that rumination contributes to the development and maintenance of depressive episodes (Just & Alloy, 1997; Nolen-Hoeksema, Morrow, & Fredrickson, 1993). Furthermore, rumination has been shown to mediate the relationships between certain risk factors (e.g., negative cognitive styles, self-criticism, and neediness) and prospective onsets of major depression (Spasojevic & Alloy, 2001). It should be noted that this chain of reasoning is speculative at this point. Still, it suggests some of the possible implications for studying sustained pupil dilation in depressed individuals and subclinical populations at-risk for depression. In this vein, Steidtmann (2006) recently investigated the relationship between pupil dilation and emotional information processing in formerly depressed individuals following a depressive mood prime. Interestingly, formerly depressed individuals in this study exhibited greater cognitive load than never depressed individuals following negative word stimuli *prior to* but not following a negative mood induction, which was seemingly inconsistent with previously mentioned priming literature.

Sustained Pupil Dilation and Dysphoria

Although currently depressed individuals have been found to exhibit greater sustained pupil dilation to emotional information, it has yet to be determined whether individuals with dysphoria (i.e., not meeting MDD criteria) also exhibit this disproportionately greater sustained processing of emotional information. Assessing sustained pupil dilation on the basis of dysphoria could help clarify whether this phenomenon occurs only at-or-above the severity threshold of major depression, or if it could be observed at less severe levels.

In order to investigate this question, the presence of dysphoria and absence of current or past depression in participants needs to be determined through a multiple gating procedure (Haaga & Solomon, 1993; Kendall & Flannery-Schroeder, 1995; Kendall et al., 1987). This ensures that participants' dysphoria status was both stable over time and present during the experiment. Multi-modal assessment has also been recommended when assessing the incidence of a past or present depressive episode (Hodgins, Dufour, & Armstrong, 2000). For instance, the clinician-administered *Structured Clinical Interview for DSM-IV* (SCID-I/NP) can increase confidence that participants who do not self-report a history of depression on the *Inventory to Diagnose Depression-Lifetime Version* (IDD-L) truly have not had a major depressive episode. By excluding participants with a history of depression, a cross-sectional methodology could establish the logical precedence of sustained pupil dilation to any eventual first onset of depression, if that effect were found. Also, restrictive BDI scoring ranges need to be utilized for assigning participants to dysphoric and

nondysphoric groups, because no prior studies have investigated whether sustained pupil dilation differences exist based on dysphoria status. Kazdin (2003) has argued persuasively that it is sensible to select conditions that maximize the likelihood of showing effects, in this case, differences between dysphoric and nondysphoric groups.

If dysphoric individuals were found to exhibit greater sustained pupil dilation than nondysphoric individuals in response to an emotional information processing task, it might suggest that cognitive elaboration of emotional information contributes to cognitive vulnerability for developing major depression. Finding such differences would also correspond with continuity theories of depression. On the other hand, failing to find differences, given adequate power, might support a noncontinuity conceptualization.

The current study examined sustained pupil dilation in dysphoric versus nondysphoric individuals in response to an emotional information processing (e.g., valence identification) task in order to ascertain whether this neurocognitive correlate of depression exists in the absence of, and therefore prior to, the development of major depression. A mixed 2 x 2 design was employed, consisting of a two-level within-subjects variable of word valence (e.g., positive, negative), and a two-level between subjects variable of dysphoria status (e.g., dysphoric, nondysphoric). Multiple gating and multimodal assessment was employed to determine the presence and stability of dysphoria, and the absence of past major depressive episodes, respectively. Participants completed the PANAS and BAI so that levels of positive

and negative affect related to dysphoria could be isolated and explored in the study's dysphoric sample.

Based on previous findings with depressed individuals, it was hypothesized that dysphoric individuals would exhibit greater sustained pupil dilation than nondysphoric individuals in response to presented negative words. It was also hypothesized that dysphoric individuals would show greater sustained pupil dilation following negative words than following positive words. Main or interaction effects involving gender have not been reported previously in this literature and were not expected in this investigation. Nonetheless, the theoretical relevance of gender to rumination (e.g., Nolen-Hoeksema, Larson, & Grayson, 1999; Nolen-Hoeksema et al., 1993) highlighted gender as a potentially important variable to examine in the context of sustained pupil dilation. Thus, gender was examined as an independent variable to be ruled out. From a continuity perspective, effects were expected to be of lesser magnitude, given the lesser severity of dysphoria compared to major depression. Thus, it was decided that statistically non-significant interaction trends would be explored further through the analysis of simple main effects.

Although certain studies of information processing have suggested that dysphoric individuals might engage their attention less with positive information than nondysphoric individuals (Siegle et al., 2001), no related pupil dilation bias has been reported in previous studies of relevant populations. Thus, this possibility was examined, though no *a priori* prediction was made. With respect to reaction times, past research influenced this study's hypothesis that dysphoric individuals would

react significantly more quickly to negative information than to positive information, in contrast to nondysphoric individuals who were expected to exhibit the opposite pattern. Lastly, exploratory analyses were planned to examine whether parental history of depression might affect participants' pupil dilation to emotionally valenced words. Because this relationship had not been examined in previous investigations, *a priori* hypotheses were not made.

Method

Data were gathered from participants in a mass-testing session as well as during an experimental session. As part of the mass-testing, potential participants completed the *Beck Depression Inventory* (BDI), the *Beck Anxiety Inventory* (BAI), the *Inventory to Diagnose Depression-Lifetime Version* (IDD-L), and the *Positive and Negative Affect Schedule* (PANAS). Initial eligibility for the experimental session was determined based on mass-testing BDI scores and IDD-L scores (see inclusion and exclusion criteria below). At the time of the experiment, potentially eligible participants completed the same series of instruments again, with the exception of the IDD-L. Participants also completed a valence identification task and the interviewer-administered *Structured Clinical Interview for DSM-IV-I, Non-patient Edition* (SCID-I/NP). All of the above-mentioned instruments, except the IDD-L and SCID-I/NP, were used in analyses. In addition, reaction times for participants' responses and sustained pupil dilation were measured and analyzed.

Participants

Sixty-two University of Kansas students (29 dysphoric, 33 nondysphoric) were recruited from the Department of Psychology mass-testing program. To balance any gender effects across dysphoria status groups, the total sub-sample of each gender were divided relatively equally between the two dysphoria status groups. Because comparing gender effects was not a central goal of the study, the overall sample was not intentionally balanced for gender. This resulted in having more females than

males *within* a dysphoria status group (e.g., 20 females, 9 males in dysphoric group). However, this imbalance was roughly matched in the other dysphoria group (e.g. 21 females, 12 males in nondysphoric group), preserving between-groups similarity.

Inclusion Criteria

Individuals were initially eligible if they met criteria for dysphoric or nondysphoric based on Beck Depression Inventory scores. Individuals scoring in the 10-17 range were selected for the dysphoria group, whereas those scoring in the 0-5 range constituted the nondysphoric control group. At the time of the experimental session, they were administered the BDI a second time, and needed to score in the same dysphoric or nondysphoric range to be eligible for the study. Participants also needed to possess sufficient English language skills to read and comprehend the word stimuli and questionnaires.

Exclusion Criteria

Individuals with a history of major depression, as indicated by the Inventory to Diagnose Depression-Lifetime Version or the Structured Clinical Interview for DSM-IV-I, Non-patient Edition, were excluded from analyses. The IDD-L was administered in the initial mass-testing to screen out some formerly depressed individuals prior to recruitment efforts. The SCID-I/NP, which was administered at the time of the experiment, served as corroboration for the lack of a past or current major depressive episode in qualifying participants. Participants were required to be free of any other current psychiatric or health disorders that could have interfered with research participation.

Stimulus Task

Valence identification task

Using a computer program (Siegle, 2000), a list (see Appendix G) of 60 words (balanced for valence, length, order, and normative frequency of usage) was presented one at a time on a computer screen. The words will consist of white, lower-case letters approximately 1.59cm high, subtending 1.4 degrees of visual angle on a black background. Participants were instructed to name the emotional valence of each word by pushing specific game pad buttons (sensitive to reaction times within milliseconds) that represent positive, negative, or neutral as quickly and accurately as possible. The positioning of the symbols (+, -, or n) was counterbalanced across participants, and labels for these responses also appear at the top right corner of the screen. Per Siegle, et al. (2001), the procedure began with the presentation of a fixation square on screen for 200 msec. A row of X's (i.e., forward mask) appeared before and immediately after the presentation of each word, which appeared on-screen for 150 msec. At this time the participant was able to respond. Pupil dilation was recorded for 12 seconds after the initial onset of the word stimulus, regardless of when the participant responded.

Measures

The Beck Depression Inventory (BDI)

The Beck Depression Inventory is among the most widely used depression self-report measures in the world and has considerable reliability and validity data to support its use. It has a high degree of convergent validity with other depression

measures, however it has less impressive discriminative validity. That is, the BDI is a sensitive measure of syndrome depression, but it is *not specific* to depression (Kendall & Flannery-Schroeder, 1995). For example, aggregated high scores on the BDI are to a lesser extent correlated with anxiety. It has been proposed that the BDI is truly measuring *dysphoria* (Beck, Steer, & Garbin, 1988), which is a core element of depression (Frank et al., 1992; Ingram & Wisnicki, 1999). For each of the 21 BDI items, four answer choices are offered. Item scores range from 0-3, with 0 reflecting the absence of a specific symptom and 3 reflecting severe and frequent presence of a symptom. encompassing a range from the absence of a particular symptom to the frequent and severe presence of the symptom. Scores on the BDI can range from 0-63, with scores below 10 suggesting nondepression, scores between 10 and 17 indicating dysphoria, and scores between 20 and 63 indicating depression (Kendall et al., 1987).

The Beck Anxiety Inventory (BAI)

The BAI (Beck, Epstein, Brown, & Steer, 1988) is a 21-item self-report instrument meant to measure current severity of anxiety symptoms, as differentiated from depressive symptoms. The BAI has demonstrated high internal consistency and test-retest reliability and satisfactory concurrent and discriminant validity (Beck, Epstein et al., 1988; Hewitt & Norton, 1993). Correlations between BAI and BDI scores tend to be moderately high, but are still significantly lower than correlations between other anxiety measures and the BDI (Beck, et al., 1988). Thus, use of the

BAI has been recommended to differentiate anxiety and depression syndromes in clinical and nonclinical populations. BAI scores can range from 0-63.

The Inventory to Diagnose Depression-Lifetime Version (IDD-L)

The IDD-L is a 24-item self-report instrument used to diagnose a lifetime history of major depressive disorder. It has been demonstrated to have sensitivity and specificity similar to the Diagnostic Interview Schedule, as well as satisfactory construct and discriminant validity (Sakado, Sato, Uehara, Sato, & Kameda, 1996; Zimmerman & Coryell, 1987) and test-retest reliability (Sato et al., 1996). The IDD-L asks the individual to consider the week in his or her life where the most depression was experienced. Each inventory item assesses a different facet of a depressive symptom on a five-point severity scale, which typically ranges from the absence to the significant presence of a symptom. IDD-L scores can range from 0-96, with scores above 40 suggesting the presence of a previous major depressive episode.

The Positive and Negative Affect Schedule (PANAS)

The PANAS is a self-report instrument consisting of two 10-item scales that measure the two primary dimensions of mood. Twenty adjectives describing various positive and negative emotions are presented, and participants rate each adjective on a five-point Likert scale based on the degree to which they are currently experiencing it. Items from each scale are summed (missing items are prorated) to compute a negative affect scale score and a positive affect scale score. Thus, the PANAS yields scores in the range of 10-50 on both the negative affect scale and the positive affect scale, with higher scores indicating stronger affect. The two PANAS scales have been

shown to be highly internally consistent, largely uncorrelated, and temporally stable (Watson, Clark, & Tellegen, 1988). The two scales also correlate appropriately with measures of related constructs. When introduced with a short-term focus (e.g. “right now” or “today”), the scales are sensitive to variations in mood.

The Visual Analog Scale (VAS)

The VAS is a relatively coarse measure in which a participant marks an “X” along a 10cm line to indicate the degree to which he or she is currently feeling sadness. The extreme ends are “not sad at all” and “very sad.” The VAS is used as a means of quickly, unobtrusively assessing mood, often multiple times within experimental designs where fluctuations in mood state must be considered.

The Structured Clinical Interview for DSM-IV-I, Non-patient Edition (SCID-I/NP)

The SCID-I/NP is a semi-structured interview used (First, Spitzer, Gibbon, & Williams, 2002) to make DSM-IV-TR diagnoses (American Psychiatric Association, 2000). The SCID consists of modules, which correspond with DSM-IV axes and classes of disorders. Each module is constructed as a schematic algorithm that leads to a diagnostic conclusion, based upon the examinee’s reporting and the clinician’s judgment. As noted earlier, the SCID will be used chiefly to rule out current and past depressive episodes. Therefore, only the unipolar and bipolar depression modules of the SCID-I/NP will be administered. The depression portion of the SCID has been shown to be reliable (Zanarini & Frankenburg, 2001; Zanarini et al., 2000).

The Ruminative Response Scale (RRS) - short form

The RRS is a self-report scale within the commonly used Response Styles Questionnaire (Nolen-Hoeksema & Morrow, 1991). The short form utilized in this study consisted of 8 factor-analyzed items that remained after items that referred overtly to depressive symptoms were removed (e.g., Roberts, Gilboa, & Gotlib, 1998). Five items load onto an *introspection/self-isolation* factor, while the other three items load onto a *self-blame* factor. Each item lists a possible response to a sad mood state and asks the respondent how frequently he or she acts this way. Item responses on the four-point Likert scale range from “almost never” to “almost always.” The complete 21-item version of the RRS has demonstrated good internal consistency (Nolen-Hoeksema & Morrow, 1991).

The Penn State Worry Questionnaire (PSWQ)

The PSWQ (Meyer, Miller, Metzger, & Borkovec, 1990) is a 16-item self-report measure of the tendency to worry, which uses a 5 point Likert scale ranging from “Not at all typical” to “Very typical.” The measure has demonstrated high internal consistency when administered to anxiety disordered and mentally healthy individuals. It has also demonstrated acceptable convergent and discriminant validity with measures of anxiety and depression, respectively (T. A. Brown, Antony, & Barlow, 1992).

The Family History Screen (FHS)

The Family History Screen (Weissman et al., 2000) is a brief interview designed to collect information relevant to 15 psychiatric disorders in informants and their first-degree relatives. The present study only assessed questions from the

depression module, for which acceptable validity for identifying depressive episodes has been demonstrated (Weissman et al., 2000). Per FHS protocol, informants were first asked whether they, their father, mother, or siblings had ever experienced symptomatic phenomena (e.g., depressed mood, sleep disturbance, anhedonia/fatigue). If a symptom was endorsed for any of the relatives being assessed, a follow up question was asked to assess whether the symptom had persisted for two weeks or longer.

Reaction Times and Sustained Pupil Dilation

A series of words was presented, one at a time, on a computer screen. Participants were instructed to identify the emotional valence (e.g., positive, negative, neutral) as quickly and as accurately as possible. The interval of sustained pupil dilation following the presentation of each stimulus was measured and analyzed. Because participants were attempting to identify word valences quickly, reaction times were collected by the valence identification task software.

Equipment

An ISCAN, Inc., Model RK-464 pupillometer, a Web camera, and an infrared light source pointed at the participants' eye was used to detect and record pupil dilation preceding and following the presentation of stimuli. Pupil size and location were recorded at 60Hz (every 16.7 msec) and were passed to a computer that controlled the display of the stimuli, as well as a computer that acquired the data. Based on the performance of pupillometers in past research, it was expected that the equipment would measure pupil diameter at .05mm resolution (Siegle, et al., 2001).

Experimental Procedure

Participants who were likely to be eligible based on their mass testing scores were invited to participate in the experiment. When they came in, participants reviewed a study consent form with a research assistant. After consenting, participants were given the BDI, BAI, PANAS and several demographics questionnaires to complete. Given that the participant's total BDI score was in the same dysphoric (10-17) or nondysphoric range (0-5) as it was during mass testing, the participant continued in the study. Those who did not score in the same range were excused and received full research participation credit for their introductory psychology course.

Prior to calibrating the eye tracker, a handheld vision test was administered. Qualifying participants needed to demonstrate 20/30 corrected vision in each eye, using a handheld eye chart (see appendix for instructions). The participant was instructed to extend a wooden eye chart apparatus from the front of the neck and to read the 20/30 vision line. Next, the eye calibration task was described to the participant (see appendix). The participant was seated in front of the eye tracker, and a chin/head rest was adjusted vertically so that his or her head remained stable and comfortable during the experiment. The brighter light in the room was turned off, leaving only a dim light on. The eye tracker was calibrated to the individual's pupil so that it followed shifts in gaze. In order to accomplish this, the individual sat in front of the computer monitor, placed his or her chin on the chin rest, and fixated on the target "X" as it appeared at various positions on the screen. The research assistant

saved the file and assessed whether the eye tracker had accurately calibrated. If not, the calibration procedure was repeated.

After successful calibration, the research assistant saved the file and instructed the participant about the subsequent valence identification task (see appendix). Participants first completed a brief practice version of the valence identification task in order become familiar with the configuration of the three response choices (positive, neutral, and negative) and the gamepad buttons. If the participant voiced a desire to practice further, the brief practice version was repeated. Otherwise, the experimental valence identification task began. This computerized task lasted approximately 12 minutes, and two automatic breaks—as long as the participant chose to take—divided the task in thirds.

Following this, an interviewer administered the current and past mood portions of the SCID-I/NP to the participant in order to rule out current or past unipolar or bipolar depression. Once these steps were completed, the participant was debriefed (see appendix) about the study and was provided an opportunity to ask questions about the rationale behind the experiment. Irrespective of participants' mood status, they were provided educational contact information about depression and community mental health resources.

Data Selection, Cleaning, and Reduction Procedures

Calculation of Pupil Dilation Indices

Pupil dilation difference indices were calculated by obtaining the average pupil diameter over the 1 second preceding the stimulus onset (i.e., baseline dilation),

then subtracting that from the average dilation following the stimulus onset. Data were cleaned with a technique previously described by Granholm, et al. (1996) and subsequently used in another investigation of information processing and pupil dilation (Siegle, et al., 2003). Eye blinks were identified as large changes in pupil dilation that occur too rapidly to represent true dilation or contraction. Word trials in which greater than half of the data points were identified as blinks or in which the participant blinked during the baseline pupil measurement (i.e., the average pupil size in the 1 second preceding stimulus onset) were excluded from a participant's analyses. In trials not characterized by excessive blinking, blinks were removed, and linear interpolations replaced them in the data set, as described in Siegle et al. (2003). Also, linear trends were calculated over the blocks of 20 trials in order to eliminate the effects of slow drift in pupil diameter that are not related to task characteristics (i.e., cognitive fatigue).

Calculation of Reaction Times

Harmonic means of reaction times were employed to indicate the central tendency of an individual's reaction times within a condition (Ratcliff, 1993). Trials with reaction times below 150 msec or over 5000 msec were removed from analyses as outliers, because previous results indicate that reaction times in these ranges are made without regard for the stimulus (Matthews & Southall, 1991). It was decided *a priori* that reaction time analyses would focus on only those trials where the participant responded in accordance with the normed valence of a word. Similarly, it

was decided that only responses to positively and negatively valenced words would be analyzed.

Analytic Strategy

For reaction times, a 2 x 2 ANOVA was planned with dysphoria status (dysphoric, nondysphoric) and valence (positive, negative) as independent variables.

Two strategies were employed to examine effects of independent variables of interest on pupil dilation. First, between-group contrasts on pupil dilation were examined at all points along pupil dilation waveforms. Based on past studies' analyses, regions of the waveforms were considered to be significantly different when more than 1.36 seconds of consecutive tests (17 data points, occurring every .08s) were statistically significant at $p < .1$ (e.g., Guthrie & Buchwald, 1991; Siegle, et al., 2003). This strategy has been shown to provide acceptable control for type I error, and was used as a primary means of identifying differences.

Secondly, to supplement and aid in additional analyses, pupil dilation data were smoothed into 250ms averages. These data were then subjected to a principal components analysis (PCA), excluding pupil responses to neutral words. PCA is a commonly used technique for analyzing psychophysiological data. In this case, 48 means from the quarter-second intervals comprising the 12-second time course for each participant were considered variables. Thus, components reflected clusters of consecutive time points with high bivariate correlations. PCA organized the data into potentially meaningful structures, which may reflect onsets of qualitatively different cognitive events (e.g., Siegle et al, 2001), and which were subjected to relevant

hypothesis tests. Analysis of variance (ANOVA) was employed to identify main effects and interactions of interest. Dysphoria status, word valence, and gender were independent variables, and regressed PCA component scores were considered dependent variables in these independent ANOVAs. ANOVA was chosen as an analytic technique that would provide conservative tests of relationships of interest and potentially greater power than a MANOVA. Ultimately, dual strategies were chosen to exploit the unique strengths and minimize the weaknesses of each approach. Requiring 17 consecutive tests to be significant at $p = .10$ rigorously controlled for spurious type I errors when comparing two groups. On the other hand, utilizing ANOVA with PCA components derived from the pupil dilation data enabled the conservative testing of interaction effects and the detection of between group differences in components (i.e., cognitive events) that may last less than 1.36 seconds. Also, where these two strategies provided overlapping results, differences could be more meaningfully and thoroughly interpreted.

Prior to examining the primary dysphoria status by word valence interaction effect, it was necessary to rule out other unexpected, possibly confounding main or interaction effects. Techniques to control potential type I error (i.e., Bonferroni correction) were intentionally not employed because these techniques increase the chance of committing a type II error, which was of greater importance in these preliminary analyses. That is, identifying a potential confounding variable would argue for including it in tests of the primary hypothesized analysis (i.e., dysphoria status by valence by *confound*).

Results

Data selection, cleaning, and reduction procedures refined the final data set to be analyzed. To begin with, the data of four nondysphoric participants were excluded from analyses because they endorsed levels of anxiety on the BAI in excess of what is considered to be the 75th percentile in community settings (Gillis, Haaga, & Ford, 1995). With respect to pupil dilation data, an average of 6.8 % of trials per participant ($SD = 5.9$ %) were excluded from further analysis due to excessive or baseline eye blinks. For the principal components analysis, promax rotation was used because the components showed evidence of being moderately correlated (i.e., non-orthogonal), and because oblique rotation produced cleaner time point variable loadings onto components.

With regard to reaction time data, removing trials shorter than 150ms or longer than 5000ms resulted in the exclusion of 0.7% of the reaction time data. Also, participant responses that were inconsistent with the normed valence of particular words (e.g., response was neutral to a word normed as positive) were identified and examined for potential group differences. A 2 (dysphoria status) x 2 (gender) ANOVA was conducted on these inconsistent responses, but no significant or main effects were found, indicating that the groups and subgroups showed relatively equivalent accuracy in their responses. Overall, participants responded consistently with the normed word valences on 85% of trials. In addition, reaction times on the valence identification task were positively skewed, skew = 1.68. This degree of skew nonetheless fell below the generally accepted cutoff absolute value of 2, indicating a

relatively normal distribution. Thus, normalization transformation techniques were not applied to the data.

Following these data refining procedures, three main sets of analyses were performed. The first set examined possible reaction time differences between dysphoric and nondysphoric individuals completing a valence identification (VID) task. The second set examined potential differences in sustained pupil dilation between dysphoric and nondysphoric individuals during the VID task. A third set of analyses compared these groups on self-report measures in order to help explain behavioral and physiological findings.

Participant Demographics and Descriptive Information

Recruitment, data selection, cleaning and smoothing resulted in analyses of 62 participants. The overall sample included 29 dysphoric and 33 nondysphoric individuals. The dysphoric group was composed of 20 females, and the nondysphoric group included 21 females. The dysphoric and nondysphoric groups did not differ significantly in terms of age, gender composition, ethnicity, or time between the prescreen and experimental sessions. After discovering an unexpected gender by dysphoria status interaction in the pupil dilation data (as described later), a 2 x 2 ANOVA was conducted to examine a possible interaction effect of gender and dysphoria status on age. Although a significant main effect of gender on age was found in the data set, $F(1, 62) = 4.55, p < .05$, no interaction between groups was found. The male group ($M = 20.00, SD = .469$) was older than the female group (M

=18.77, $SD = .332$), but this effect was shared relatively equally across the dysphoric and nondysphoric groups.

Mood and Affect

The dysphoric group's mean experimental BDI score ($M = 12.07$, $SD = 2.56$) did not differ significantly from its mean prescreen BDI score ($M = 12.18$, $SD = 2.91$), supporting a moderate degree of stability across the intervening time period ($M = 45.17$ days, $SD = 18.89$)¹. Examining data from item #1 on the BDI, which assesses self-reported sadness, the dysphoric group's modal and median response was a score of 1 (e.g., "I feel sad"). However the mean score for the group was only 0.66 ($SD = 0.48$), uncovering the fact that 10 participants had endorsed a score of zero (e.g., "I do not feel sad") and highlighting that the overall sample was characterized by relatively mild sadness. Because the study examined group comparisons in reaction times on trials that required rapid decision making, group mean responses to BDI item #13 (i.e., assesses self-reported indecisiveness) were compared. Dysphoric individuals endorsed greater indecisiveness² ($M = 0.90$, $SD = 0.72$) than nondysphoric individuals ($M = 0.09$, $SD = 0.38$), $t(41.36) = 5.36$, $p < .001$. However, neither gender nor gender by dysphoria status effects were found. Additionally, as expected the dysphoric group endorsed significantly higher levels of anxiety on the BAI than the nondysphoric group, $t(32.95) = 8.10$, $p < .001$ (equal variances not assumed). In fact, the dysphoric group's mean elevated anxiety score (see Table 1 for means and standard deviations) fell in the 80-90th percentile range of what may be characteristic in community settings (Gillis et al., 1995).

The dysphoric group also endorsed higher levels of negative affect on the PANAS, $t(60) = 5.72, p < .001$ than the nondysphoric group. Although the nondysphoric group endorsed marginally greater positive affect than the dysphoric group, this difference did not reach statistical significance. Examination of data from the visual analog mood scale corroborated that the dysphoric group was experiencing a significantly greater degree of sadness than their nondysphoric counterparts before and after completing the VID task, $t(41.45) = 5.38, p < .001$, and $t(41.04) = 4.79, p < .001$. The dysphoric group marked an “X” at a mean distance of 3.1 cm from “not sad at all” toward “very sad”, whereas the nondysphoric group averaged only 0.8 cm along this dimension.

Rumination and Worry

As predicted, the dysphoric group endorsed greater tendencies toward rumination and worry than the nondysphoric group. The mean scores for the dysphoric group were significantly higher than the nondysphoric group mean on the short form RRS, $t(59) = 5.39, p < .001$ and on the PSWQ, $t(58) = 8.56, p < .001$. Again, the dysphoric group mean on the PSWQ fell in the 80-90th percentile range of another normative community sample (Gillis et al., 1995). Two 2 (dysphoria status: dysphoric, nondysphoric) x 2 (gender: female, male) ANOVAs were conducted with the RRS and PSWQ data, respectively. The main effect of dysphoria status was confirmed for both the RRS [$F(1, 57) = 33.98, p < .001$] and PSWQ [$F(1, 57) = 55.97, p < .001$], although the rumination main effect was qualified by a trend-significant interaction, $F(1,57) = 3.82, p = .06$. Follow up tests revealed that

nondysphoric females endorsed significantly higher levels of rumination ($M = 13.81$, $SD = 0.83$) than nondysphoric males ($M = 10.73$, $SD = 1.14$), $F(1, 57) = 4.80$, $p = .03$. However, dysphoric males ($M = 18.78$, $SD = 1.26$) and dysphoric females ($M = 17.82$, $SD = 0.85$) endorsed similarly elevated levels of rumination.

Parental History of Depression

Data from the Family History Screen were collapsed into a binary variable consisting of “no” or “yes” regarding the presence of family history of depression. This was done so that data could be analyzed in pairwise comparisons. It was decided that only data regarding parental history of depression would be utilized to determine which participants qualified as “high risk” versus “low risk.” This was done partly in order to control for disproportional probability introduced by the variability of the number of first-degree relatives. For example, a participant with four siblings would necessarily be more likely than an only child to be in the high-risk group (i.e., a first-degree relative who has experienced depression), all other things being equal. Also, participants often reported less confidence in assessing past depressive symptoms in siblings than in parents, which evoked concerns about reliability of sibling reports. Overall, 27 participants, or 44.26% of the sample, endorsed items indicating that at least one biological parent had experienced an MDE. Within the dysphoric group this percentage (53.37%) was higher than in the nondysphoric group (36.36%). Also, within the dysphoric group, a greater percentage of males endorsed having a parent who had experienced an MDE (75%)³ compared to females (45%).

Table 1.

Descriptive Characteristics of Participants+

	Dysphoric	Nondysphoric	All Participants
N	29	33	62
Age in years <i>M (SD)</i>	18.86 (1.25)	19.52 (2.74)	19.21 (2.18)
Female	69.0%	63.6%	66.1%
Prescreen BDI score <i>M (SD)</i>	12.18 (2.91)***	1.28 (1.59)***	6.46 (5.95)
Experimental session BDI score <i>M (SD)</i>	12.07 (2.56)***	1.88 (2.01)***	6.65 (5.61)
BAI score <i>M (SD)</i>	15.46 (8.26)***	2.48 (2.62)***	8.55 (8.81)
PANAS Negative Affect <i>M (SD)</i>	16.03 (4.40)***	11.33 (1.61)***	25.35 (7.50)
PANAS Positive Affect <i>M (SD)</i>	24.79 (6.74)	25.85 (8.18)	13.53 (3.98)
RRS <i>M (SD)</i> +	18.11 (4.48)***	12.75 (3.24)***	15.30 (4.70)
PSWQ <i>M (SD)</i> ++	57.05 (9.83)***	26.22 (9.02) ***	46.29 (14.05)
FHS report of depression+	53.37%	36.36%	44.26%
African American/Black	-	3.0%	1.6%
Asian American/ Pacific Islander	3.4%	3.0%	3.2%
Caucasian American/White	89.7%	90.9%	90.3%
Hispanic American/ American Indian	3.4%	3.0%	3.2%
Multiracial	3.4%	-	1.6%

+Based on n = 61 because of missing data

++Based on n = 60 because of missing data

* $p < .05$, ** $p < .01$, *** $p < .001$

Reaction Time

Primary analyses

Preliminary analyses suggested that gender should be included as an independent variable. Thus, a 2 (dysphoria status: dysphoric, nondysphoric) x 2 (word valence: positive, negative) x 2 (gender: female, male) ANOVA was conducted with reaction time as the dependent variable. Significant main effects for gender $F(1, 2184) = 10.66, p = .001$, dysphoria status, $F(1, 2184) = 9.79, p = .002$, and valence, $F(1, 2184) = 3.37, p = .05$, were found. Means are listed in Table 2. Regarding valence, participants were quicker to identify negative words than positive words. Regarding gender, males responded more quickly than females to emotional words. Concerning dysphoria status, dysphoric individuals responded more quickly to emotional words than nondysphoric individuals.

As noted, a significant gender by dysphoria status interaction qualified the individual main effects of these variables, $F(1, 2184) = 29.03, p < .001$. Follow up tests for simple main effects revealed significantly different reaction times when comparing dysphoric males and nondysphoric males, $F(1, 2184) = 26.98, p < .001$, as well as when comparing dysphoric females and nondysphoric females $F(1, 2184) = 3.89, p < .05$. Dysphoric males responded more quickly to emotional words than nondysphoric males. In contrast, *nondysphoric* females identified emotionally valenced words more quickly than dysphoric females. Also dysphoric males exhibited significantly shorter reaction times than dysphoric females, $F(1, 2184) = 33.34, p <$

.001. A non-significant trend suggested that nondysphoric males exhibited *slower* reaction times than nondysphoric females ($p = .11$) following valenced words.

No other interaction effects were statistically significant. A dysphoria status by valence effect had been predicted. Specifically, it had been hypothesized that dysphoric individuals would exhibit significantly shorter reaction times than nondysphoric individuals following negative word presentations. This interaction effect was not found.

Table 2

Mean Reaction Times for the Valence Identification Task in Seconds

	Positive		Negative		Total	
	Mean	SD	Mean	SD	Mean	SD
All	1.40	.62	1.36	.57	1.38	.59
Dysphoric (all)	1.39	.64	1.33	.58	1.36	.61
Nondysphoric (all)	1.40	.60	1.38	.56	1.39	.58
Females (all)	1.41	.59	1.39	.57	1.40	.58
Males (all)	1.38	.68	1.29	.56	1.33	.62
Dysphoric Females	1.44	.61	1.42	.57	1.43	.59
Dysphoric Males	1.27	.70	1.13	.56	1.19	.63
Nondysphoric Females	1.37	.56	1.37	.57	1.37	.57
Nondysphoric Males	1.45	.65	1.40	.54	1.43	.59

Exploratory analyses

Given the marginal main effect of valence, it was decided that individual group (e.g., dysphoric) and subgroup (e.g., male dysphoric) reaction times to negative

versus positive words would be compared to determine whether the marginally significant result was driven by one or more subgroups. Visual inspection of the reaction time means, (see table 2) suggested that males responded more quickly to negative words than to positive words. A one-way ANOVA with valence as the independent variable and reaction time as the dependent variable was conducted, using data from males. Males showed a marginally significant trend toward faster reaction times to negative than positive words, $F(1, 731) = 3.47, p = .06$. Again, it was decided to examine whether this effect was disproportionately driven by the dysphoric male group, as suggested by visual inspection of the subgroup reaction time means. A one-way ANOVA with valence as the independent variable and reaction time as the dependent variable was conducted, using data from dysphoric males. Next a one-way ANOVA was conducted using only data from nondysphoric males. A trend main effect supported the notion that dysphoric males were quicker to respond to negative than positive words, $F(1, 304) = 3.68, p = .06$. However, the effect for nondysphoric males did not remotely approach significance, $F(1, 426) = 0.64, p = .42$, suggesting it could easily have been obtained through chance. Similarly, in the overall dysphoric group, dysphoric females and nondysphoric females failed to show significantly different reaction times to negative than positive words. Thus, the only simple main effect to approach significance and be consistent with the overall main effect of valence resided in the dysphoric male group.

Pupil Dilation

As previously discussed, series of consecutively significant data points were examined, in addition to ANOVAs using components derived from a PCA of the pupil dilation data. Preliminary, primary, then exploratory analyses were performed.

Component structure

Six components with eigenvalues exceeding 1.6 were extracted from the PCA of the pupil dilation data and accounted for 93.0% of the overall variance.⁴

Components are labeled in ascending order of the magnitude of variance for which they accounted and not their temporal order (i.e., first component accounted for the greatest amount of variance, but occurred fourth in the time course). It is worth noting that pupil dilation is thought to lag by 300-500ms following a cognitive event.

Reviewing the components in temporal order of the time course, the sixth component spanned 0-.75s, and may have represented pre-attentive or preparatory processing (Jennings, van der Molen, & Steinhauer, 1998). The fourth component followed spanning from .75-2.25s, and may have reflected early attentional processes, (Semmlow & Stark, 1973). Next, the third component spanned 2.25-4.75s, and may have been related to stimulus identification (G. G. Brown et al., 1999) and motor processes associated with a response (Hyönä, Tammola, & Alaja, 1995). The first component spanned 4.75-8.0s, and may have reflected associations with the response and early elaborative processing (e.g., Siegle et al., 2001). The fifth component spanned 8-10s. Although this component may have included some sustained attentional processing, its small accounting of variance (3%) suggests it might reflect a relatively universal transitional period leading into the second component, which

spanned 10-12s. The second component's relatively large accounting of variance (16%) and temporal distance from the stimulus or response suggests it may have reflected a diverse mix of continued elaborative attentional processes or disengagement of/shifts in attention among participants.

Table 3
Component Structure for the Valence Identification Task

Order in Time course	Component Label	Approximate latency to peak loading from stimulus onset (sec)	Variance accounted for as percentage
1	6 th	0.5	3.37%
2	4 th	1.5	6.36%
3	3 rd	3.5	11.21%
4	1 st	6.5	52.35%
5	5 th	9	3.44%
6	2 nd	11	16.27%
Total			93.00%

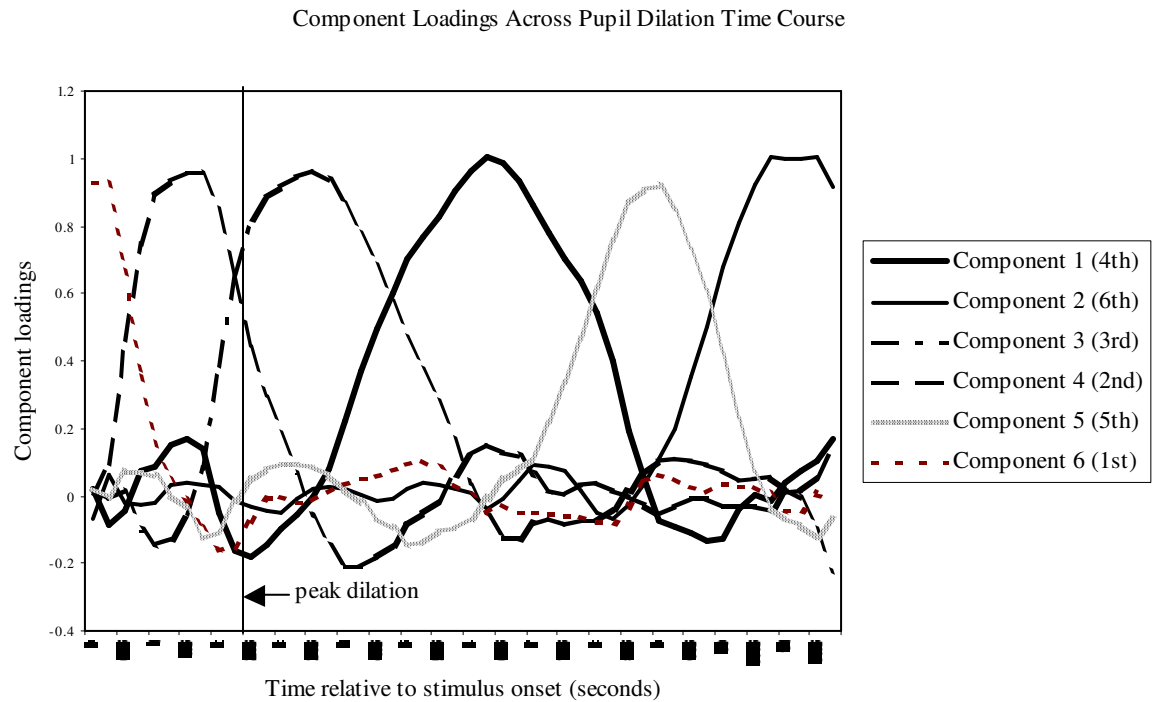


Figure 1. Component loadings for each of the six extracted components from a principal components analysis. Components are numbered in the order of magnitude of variance for which they accounted. In parentheses is the temporal order in which each component's peak occurs. Each component is plotted in a unique style. The x-axis represents the timepoints for which component loadings were derived. The y-axis represents the magnitude of the component loading.

Preliminary Analyses

Although previous studies examining pupil dilation in depressed individuals have not reported gender effects, these studies have suggested theoretical links between sustained pupil dilation and rumination, a cognitive process thought to occur more often in females. Thus, a 2 (dysphoria status: dysphoric, nondysphoric) x 2 (valence: positive, negative) x 2 (gender: male, female) ANOVA was conducted with regressed component scores for each of the six extracted PCA components, which corresponded to six timespans within the overall 12-second time course following

each stimulus presentation. Preliminary analyses examined all main effects and interactions, except for the dysphoria status by valence interaction, which is described in the primary analyses.

To begin with, a main effect of valence was apparent. The overall sample showed greater constriction following positive than negative words (below the baseline pupil dilation) from 10-12s after stimulus onset, $t(122) = 2.20, p = .03, d = -.34$. Though statistically significant, this effect was small and qualified by an interaction with gender, which is explained later. Additionally a small though statistically significant main effect of gender occurred during the third component (also third in temporal order) from roughly 2-5 seconds, $F(1, 116) = 5.03, p = .03, d = .20$. That is, females' peak dilation, collapsing across word valence levels (e.g. positive, negative), was greater than that of males. The main effect for dysphoria status did not meet criteria for significance at any point throughout the timecourse. That is, there was no apparent difference between dysphoric and nondysphoric individuals when collapsing across levels of gender and valence.

As referenced earlier, trend gender by valence effects were found. Guthrie and Buchwald's technique revealed two adjacent periods following negative words in which females showed significantly greater dilation than males [from 1.78- 2.50s: $t(60) = 1.98, p = .05, d = .53$; from 2.55 to 3.98s: $t(60) = 2.12, p = .04, d = .57$]. This is illustrated in figure 2. Gender by valence trend effects were also found in the latter two components [5th: $F(1, 116) = 2.38, p = .12$; 2nd: $F(1, 116) = 3.60, p = .06$], and within these interactions significant simple main effects were found. Specifically,

males exhibited greater dilation to positive words than females during the fifth component, $F(1,116) = 4.80, p=.03, d = .60$. This moderate-to-strong effect is depicted in figure 3. Also females showed greater dilation following negative compared to positive words in the second (last) component, $F(1, 116) = 9.38, p < .01, d = .68$ (see figure 3). Males evinced no significant differences in dilation between negative and positive words at any point throughout the time course. Thus, the previously reported main effect of word valence in the last component (e.g., #2) was driven by the tendency for *females* to dilate more following negative words than positive words.

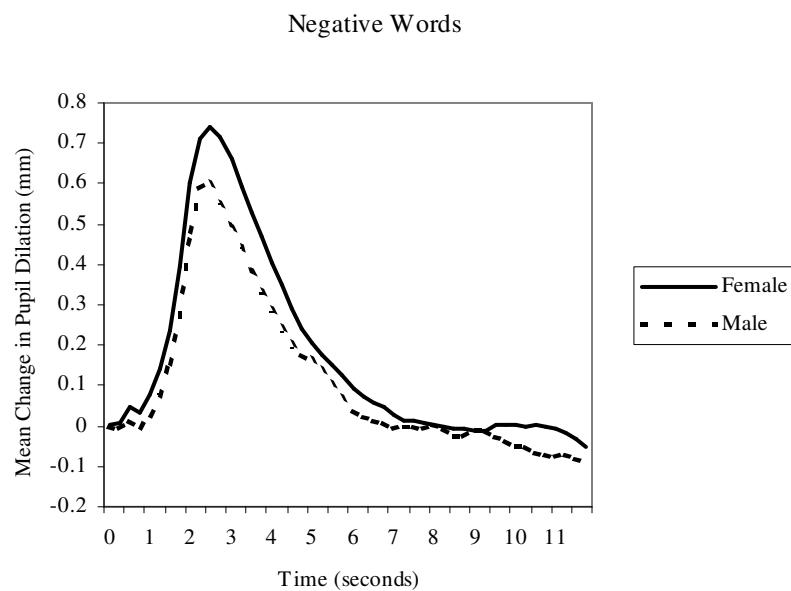
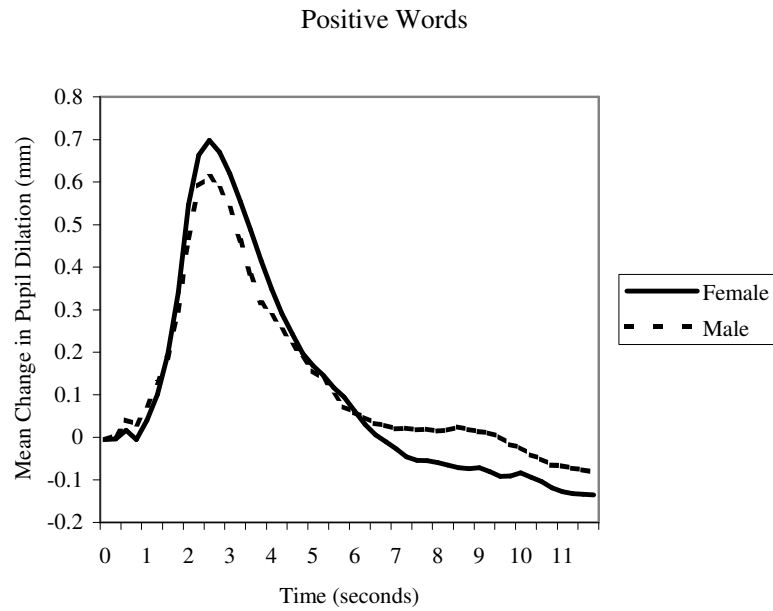


Figure 2. Mean pupil dilation comparison between female and male groups following positive words.



Figures 3. Mean pupil dilation comparison between female and male groups following positive words.

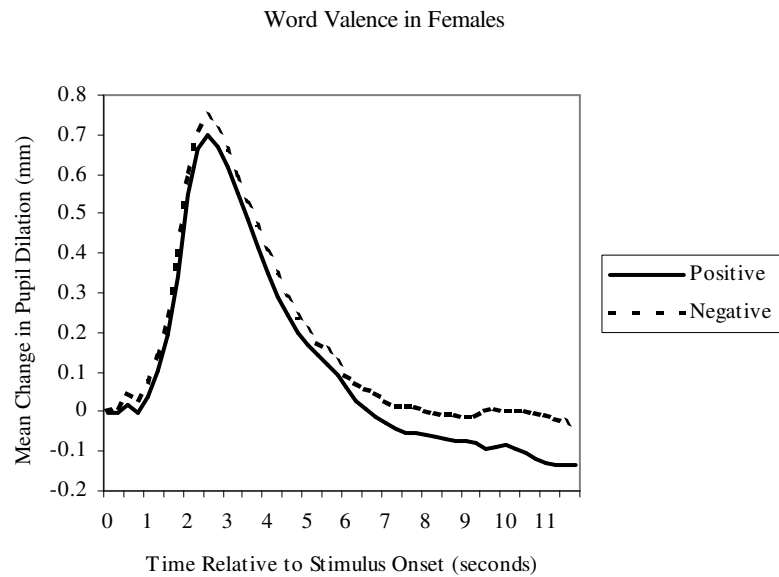
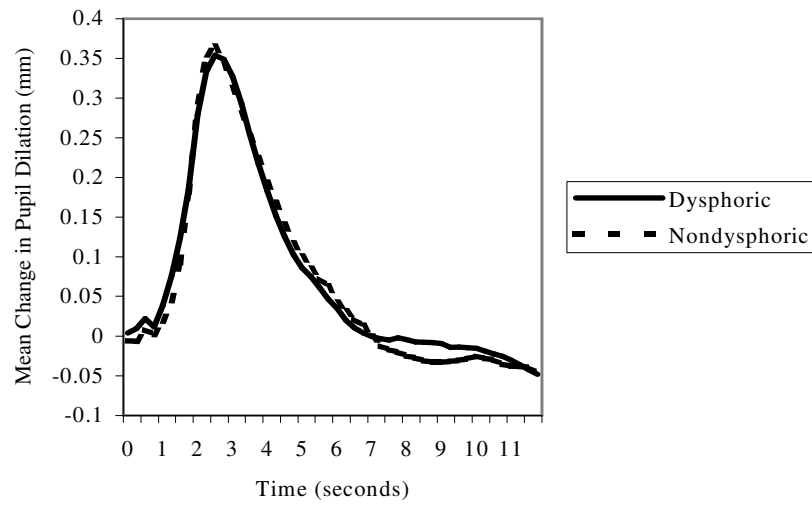


Figure 4. Within female group, mean pupil dilation following positive compared to negative word stimuli.

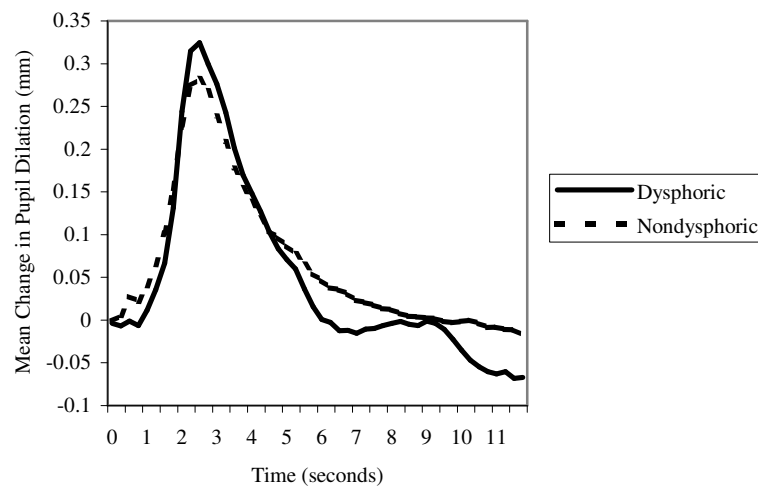
Evidence for a gender by dysphoria status interaction was also found. On the early sixth component (0-.75s post-stimulus) a significant interaction was present, $F(1,116) = 6.96, p < .01$. Follow up tests confirmed that dysphoric females exhibited greater dilation than nondysphoric females on this component, collapsing across the two valence levels, $F(1,116) = 6.03, p = .02, d = .55$, whereas dysphoric males showed a trend toward *lesser* dilation than nondysphoric males, $F(1, 116) = 2.23, p = .14, d = -.48$. These medium effect sizes are shown in figures 5 and 6, respectively. To further elucidate the interaction, trend effects are reported. Dysphoric females showed marginally greater dilation than dysphoric males $F(1, 116) = 3.37, p = .07, d = .53$, whereas nondysphoric females showed marginally lesser dilation than nondysphoric males in the initial component, $F(1, 116) = 3.62, p = .06, d = -.49$ (see figures 7, 8). During the second component (last in the time course) a trend interaction effect of dysphoria status and gender was found, $F(1, 116) = 3.23, p = .08$. Follow up tests revealed a significant simple main effect, where nondysphoric males showed greater dilation than dysphoric males, $F(1,116) = 4.28, p = .04, d = .66$ (see figure 5). Simple main effects and other relevant findings from the analysis of series of consecutive tests (e.g., Guthrie & Buchwald, 1991) are reported in exploratory analyses below.

Gender by Dysphoria Status Interaction - Females



(5)

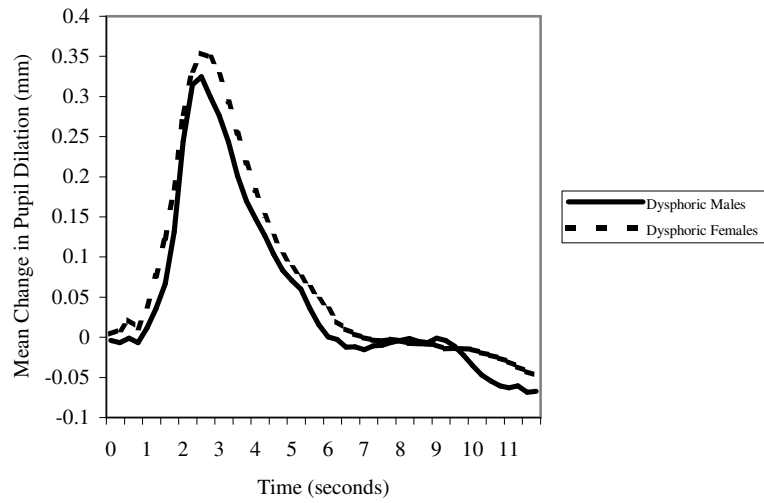
Gender by Dysphoria Status Interaction - Males



(6)

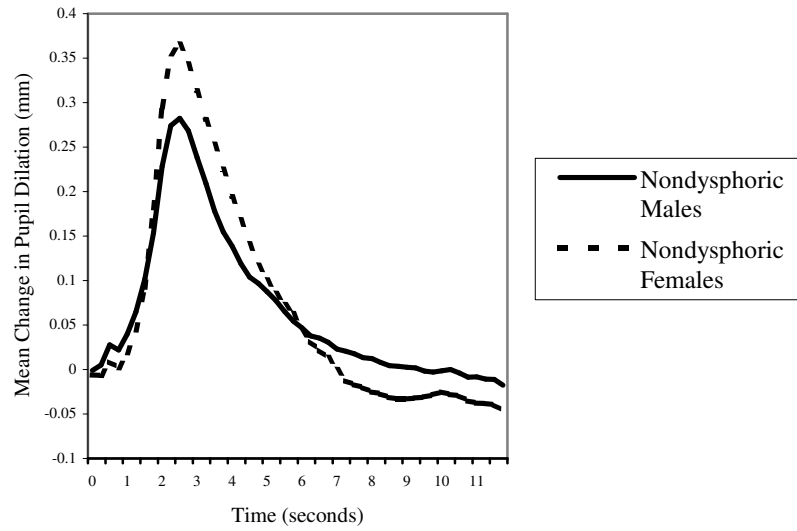
Figures 5, 6. Gender by dysphoria status interaction. Figure 5 depicts mean pupil dilation within the female group, comparing by dysphoria status. Figure 6 depicts mean pupil dilation within the male group, comparing by dysphoria status.

Gender x Dysphoria - Dysphoric Groups



(7)

Gender x Dysphoria - Nondysphoric Groups



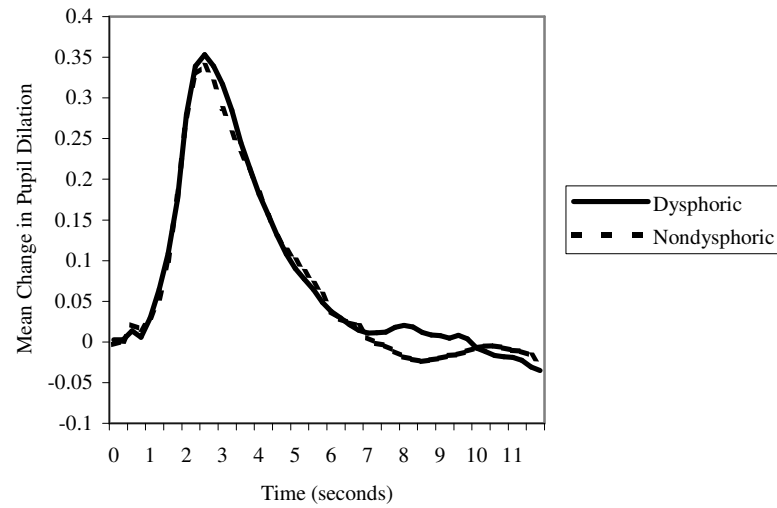
(8)

Figures 7, 8. Figure 7 depicts mean change in pupil dilation within the dysphoric group, comparing males to females. Figure 8 depicts mean change in pupil dilation within the nondysphoric group, comparing males to females.

Primary Analyses

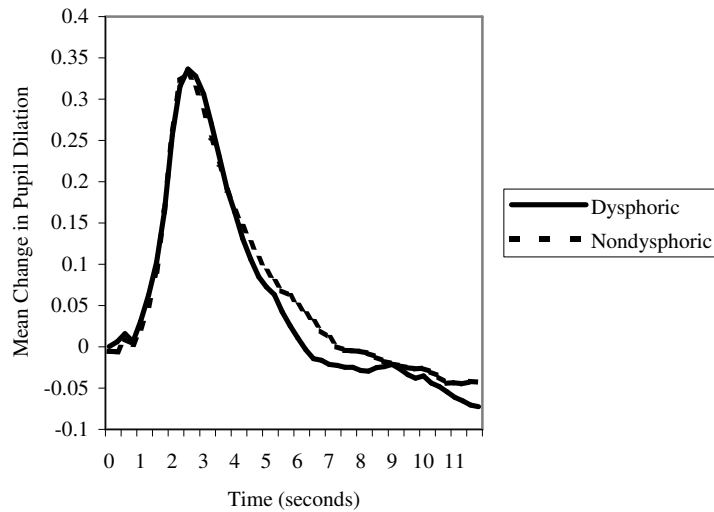
It was hypothesized that dysphoric individuals would show greater sustained pupil dilation than nondysphoric individuals following the presentation of negative words. It was also hypothesized that dysphoric individuals would show greater sustained pupil dilation after negative compared to positive words. That is, a dysphoria status by word valence effect was expected. These hypotheses were not supported. However, during the fifth component, a minimally trending interaction effect was found, $F(1,116) = 1.78, p=.19$. Results from follow up tests suggested that dysphoric individuals may show slightly greater dilation than nondysphoric individuals following negative words during the fifth component, $F(1,116) = 2.22, p=.14, d = .39$ (see figure 9), and that dysphoric individuals may exhibit slightly greater dilation to negative than positive words during the final component, $F(1, 116) = 1.88, p=.17, d =.37$ (see figure 11). Though *a priori* hypotheses were not made with regard to dysphoria status group comparisons of dilation following positive words, a nonsignificant trend suggested that nondysphoric individuals may exhibit slightly greater dilation than dysphoric individuals during the second (i.e., last) component, $F(1, 116) = 1.95, p=.17, d =.36$, as shown in figure 10. Although these non-significant trend findings should be interpreted with strong caution, these findings, along with the previously reported gender by dysphoria and gender by valence interaction effects, indicated that a gender by dysphoria status by word valence interaction should be examined.

Dysphoria Status x Valence - Negative Words



(9)

Dysphoria Status x Valence - Positive Words



(10)

Figures 9, 10. Mean change in pupil dilation following specific word valences, comparing nondysphoric and dysphoric groups. Figure 9 shows comparative pupil dilation following negative words, whereas figure 10 depicts comparative pupil dilation following positive words.

Dysphoria x Valence - Dysphoric Individuals

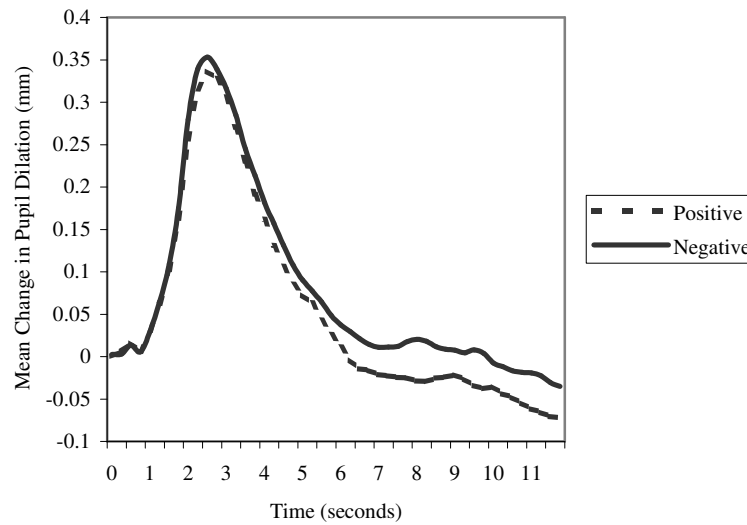


Figure 11. Mean pupil dilation within dysphoric group following positive versus negative word stimuli.

Exploratory Analyses

In order to understand the data better a set of exploratory analyses was conducted. Guthrie and Buchwald's consecutive tests technique was applied to the eight possible contrasts among dysphoria status, gender, and valence, and ANOVA follow up tests were used to examine significant simple main effects that might be embedded in an overall non-significant interaction term. Lastly, visual graphical analysis was employed to fill in explanatory gaps between the reported significant statistics.

In comparing nondysphoric males and dysphoric males, it was found that nondysphoric males sustained pupil dilation for longer than dysphoric males

following the presentation of *positive* word stimuli, particularly late in the time course (10-12s), $t(19)=3.00$, $p=.01$, $d = 1.32$ This difference is shown in figure 12.

Comparisons of nondysphoric females and dysphoric females revealed that nondysphoric females showed lesser dilation (in fact, they first constricted) immediately following the presentation of positive words (e.g., sixth component) compared to dysphoric females, $F(1, 116) = 5.68$, $p=.019$, $d = -.76$ (figure 13). This was an unexpected result. Although dysphoric females might have been expected to exhibit greater dilation than nondysphoric females following negative words, only a non-significant trend was found for this effect, $F(1, 116) = 2.88$, $p=.09$, $d = .54$, during the fifth (second to last) component (see figure 14). Comparisons of dysphoric females and dysphoric males also showed a lack of significant differences in sustained pupil dilation following words of any valence. Given preliminary results implicating gender and increased pupil dilation to negative words, it might have been expected that dysphoric females would show greater dilation than dysphoric males following negative words, but this was largely unsupported. Only slight trends in the predicted direction appeared at the very beginning (sixth component), $F(1, 116) = 2.93$, $p=.09$, $d = .71$ and end of the time course (second component), $F(1, 116) = 1.94$, $p = .17$, $d = .58$ (see figure 15).

Male Groups' Dilation to Positive Words by Dysphoria Status

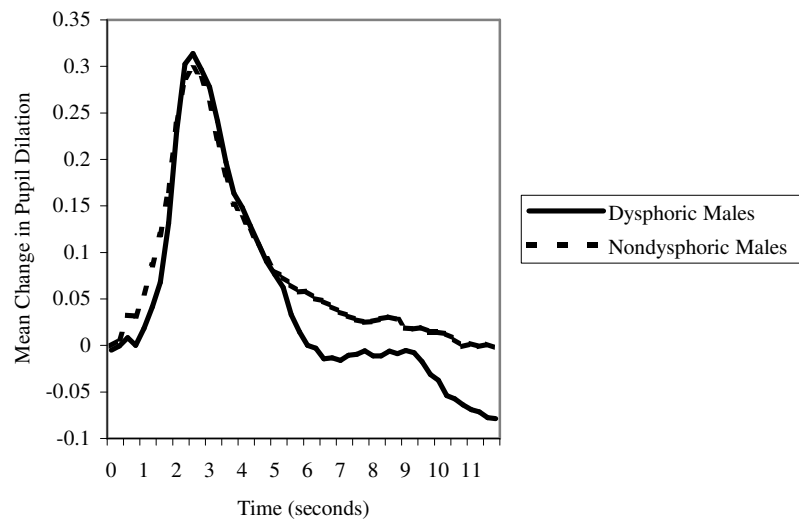


Figure 12. Mean pupil dilation following positive word stimuli within male group, comparing by dysphoria status.

Female Groups' Dilation to Positive Words by Dysphoria Status

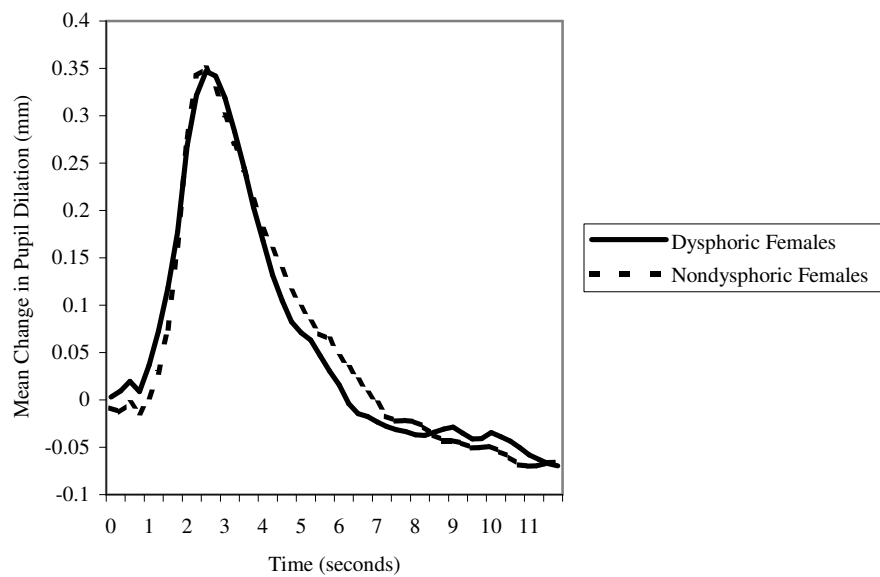
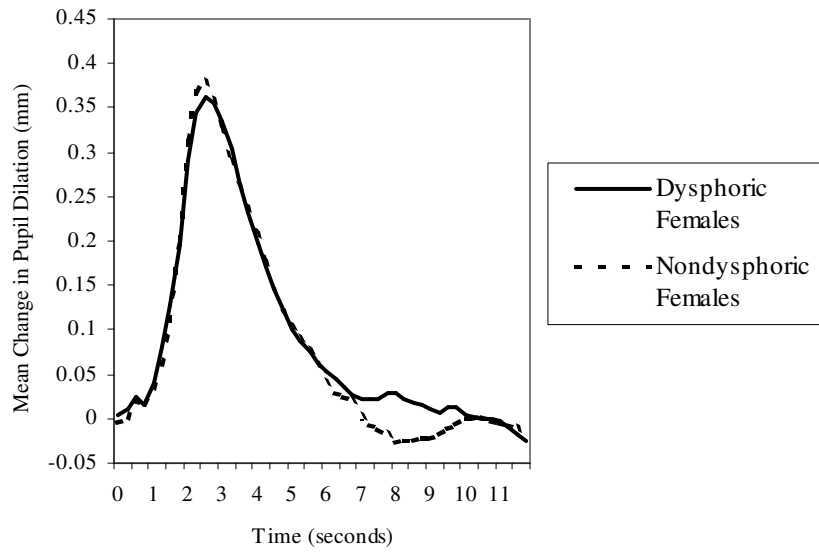


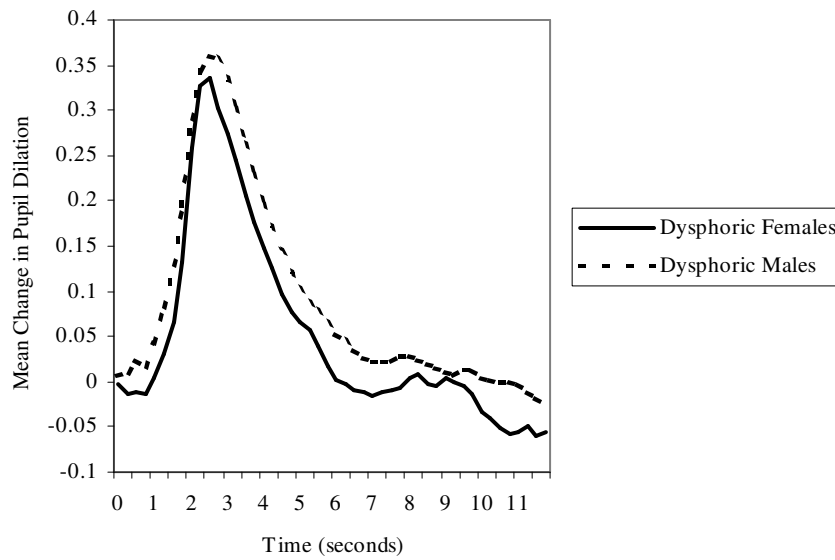
Figure 13. Mean pupil dilation following positive word stimuli within female group, comparing by dysphoria status.

Female Groups' Dilation to Negative Words by Dysphoria Status



(14)

Dysphoric Groups' Dilation to Negative Words by Gender



(15)

Figure 14, 15. Mean dilation following negative word stimuli. Figure 14 depicts dysphoric female vs. nondysphoric female comparison. Figure 15 depicts dysphoric female vs. dysphoric male comparison.

To complete the examination of possible between-group simple main effects that might contribute to the previously reported gender by dysphoria status by valence interaction, nondysphoric females were compared to nondysphoric males. It was found that following negative words, nondysphoric females showed greater dilation from 2.08 to 3.98s, including peak dilation, $t(31) = 2.13$, $p = .04$, $d = .77$. This is depicted in figure 16. Following positive words late in the time course (i.e., 8.17-12.00s), nondysphoric males showed significantly greater sustained pupil dilation than nondysphoric females, $t(31) = -2.80$, $p = .01$, $d = -1.01$ (see figure 17). Thus, the late sustained dilation following positive words exhibited by nondysphoric males distinguished them from dysphoric males, dysphoric females and nondysphoric females.

Looking within each of these four subgroups individually, comparisons of word valence (e.g. negative vs. positive) revealed differences consistent with the aforementioned gender by valence interaction. That is, females showed significantly greater late constriction following positive words than negative words, and males did not show differential dilation based on word valence. Comparisons of the effect sizes of nondysphoric females and dysphoric females revealed a lack of significant differences, indicating dysphoria status had no impact on the gender by valence effect. Also, original hypotheses would have presumed that dysphoric males would exhibit greater sustained dilation than nondysphoric males following negative words,

and greater dilation following negative words than following positive words later in the time course (e.g. fifth or second components). But these ideas were not supported.

Nondysphoric Groups' Dilation to Negative Words

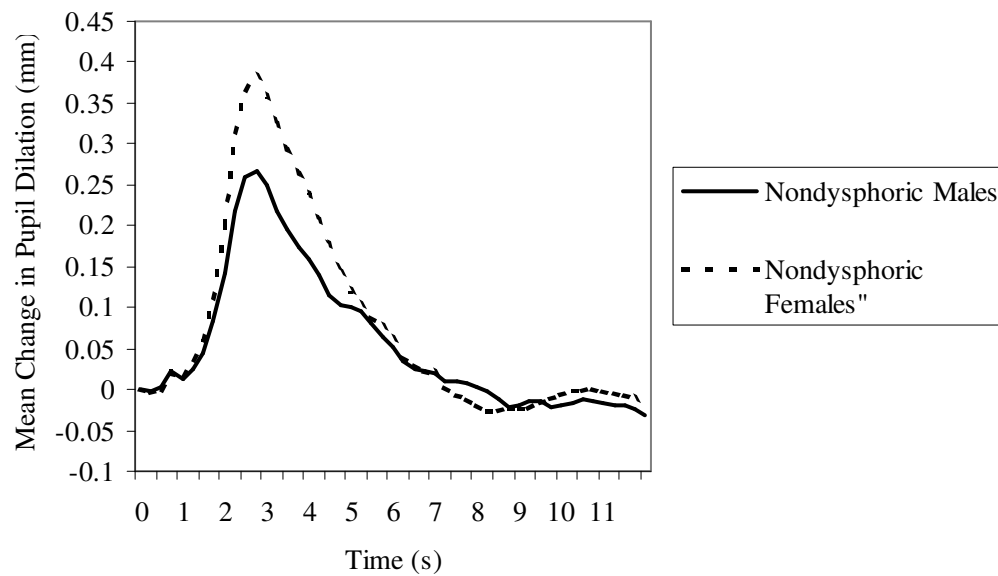


Figure 16. Mean change in pupil dilation within nondysphoric group, comparing males versus females based on negative word stimuli.

Nondysphoric Groups' Dilation to Positive Words

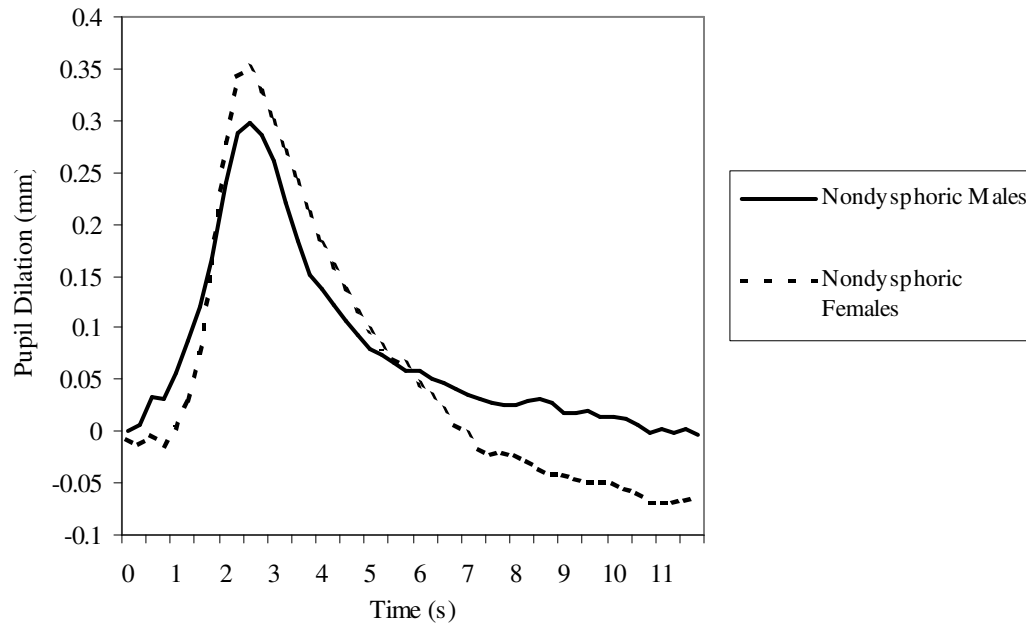


Figure 17. Mean change in pupil dilation within nondysphoric group, comparing males versus females based on positive word stimuli.

Lastly, relationships between participants' parental risk for depression and biases in sustained pupil dilation to valenced words were examined. A series of 2 (family risk: high risk, low risk) x 2 (valence: positive, negative) x 2 (dysphoria status) x 2 (gender) ANOVAs were run with each pupil dilation component as a dependent variable. Significant interactions of parental risk and valence (i.e., including three- and four-way interactions with gender and dysphoria status added in) were not found for any of the components in the pupil dilation time course. This suggests that parental risk status did not contribute to biases in pupil dilation toward positive or negative words.

Discussion

The purpose of the present study was to compare the physiological reactivity and behavioral performance of dysphoric and nondysphoric individuals on an emotional information processing task. Pupil dilation and reaction times were the main dependent variables of interest, and participant responses to psychological measures were utilized to help interpret behavioral and physiological findings.

Demographic and Psychological Findings

The dysphoric and nondysphoric groups were found to be relatively equivalent in age, but there was a significant gender effect on age in that the male group was on average a year older than the female group. Because the proportion of males was not significantly different in the dysphoric and nondysphoric groups, this did not affect interpretation of the primary pupil dilation and reaction time analyses. With regard to subsequently discussed gender effects in pupil dilation and reaction time analyses, the one-year age difference seems unlikely to constitute a confounding variable.

Findings from mood and affect measures were generally in accordance with predictions. The dysphoric group's two BDI scores were relatively equivalent, implying moderate stability of dysphoria across the average 6-week intervening period. BDI data also indicated that the dysphoric group was, on the whole, experiencing relatively mild sadness, with a third of participants endorsing that they had not felt sad across the previous two weeks. Also, compared to the nondysphoric group, the dysphoric group exhibited considerably higher levels of anxiety on the

BAI. The dysphoric group's mean level of anxiety fell in the 80-90th percentile range of what may be characteristic in community settings (Gillis, Haaga, & Ford, 1995). This is not surprising, given the high co-occurrence of depression and anxiety symptoms. Similarly, the dysphoric group endorsed significantly higher levels of negative affect on the PANAS than the nondysphoric group.

Contrary to expectations, the levels of positive affect endorsed by the dysphoric group and dysphoric group were roughly equivalent. This might suggest that reduced positive affect, as described by the tripartite model of depression (Clark & Watson, 1991), may not emerge as a distinguishing characteristic at subclinically elevated levels of depressive symptoms. Another possibility is that this sample of dysphoric college students was not characterized by predominantly depressive symptoms, but by a more balanced mixture of depressive and anxious symptoms, and thus, significantly reduced positive affect did not emerge. The VAS data provided supplemental support that the dysphoric group was experiencing significantly greater sadness than the nondysphoric group at the time of the experimental task. However, the mean dysphoric participant response was still notably closer to "not sad at all" than to "very sad."

As predicted, on the short form RRS and PSWQ the dysphoric group endorsed greater tendencies to ruminate and worry than the nondysphoric group. The dysphoric group's mean PSWQ score was considerably elevated in comparison to data from a large normative community sample (Gillis et al., 1995), again supporting the idea that this dysphoric sample was elevated in symptoms of anxiety and depression. Also, a

dysphoria status by gender effect revealed that within the nondysphoric group, females endorsed significantly greater rumination tendencies than males, but within the dysphoric group, gender differences were not found. Research by Nolen-Hoeksema and colleagues (1999; 1993) has suggested that women have a greater tendency to engage in ruminative coping than men, in the presence of, but not necessarily in the absence of, depressed moods. In the present study, mood was not manipulated, and measures of mood state preceding and following the VID task indicated that nondysphoric individuals were not experiencing depressed mood. Examination of the FHS data suggested that family history of depression was more common within the dysphoric group than the nondysphoric group.

Reaction Time Findings

The overall accuracy rate indicated sufficient engagement with and effective completion of the VID task, and differential accuracy rates among groups were not found. The primary hypothesis, that dysphoric individuals would identify negative words more quickly than nondysphoric individuals, was not supported. Also, although dysphoric individuals more quickly identified negative words than positive words, this pattern was found to some extent in nondysphoric individuals as well. The latter pattern in nondysphoric individuals contrasted with Siegle and colleagues' (2001) finding that nondysphoric, nondepressed individuals more quickly identified positive words than negative words. It is thought that reaction time data reflects the amount of effort an individual allots to information (Massaro, 1988, as cited in Siegle, Ingram et al., 2002). Related to this, in the present study, participants responded more

consistently with negatively normed words than positively normed words, perhaps suggesting that with relatively greater certitude, less cognitive effort was needed to execute intentional associative processing, response consideration, and response selection. Thus, based on the planned analyses, faster reaction times to negative words than positive words did not appear to represent a distinguishing vulnerability factor.

The lack of an interaction between dysphoria status and word valence contrasted with Siegle and colleagues' (2002) "affective interference" findings. This may be because the dysphoric sample in the Siegle study endorsed a considerably higher mean level of dysphoria. On the other hand, exploratory analyses provided some evidence that dysphoric males contributed disproportionately to the overall main effect of valence (i.e., faster RTs to negative than positive words), and that the lack of contrasting effects in nondysphoric males and nondysphoric females (i.e., faster RTs to positive than negative) diminished the possibility of a significant dysphoria by valence interaction. Although this effect was only trending and not obtained through orthodox statistical protocol (i.e., examining simple main effects only when an ANOVA interaction is significant), it is worth noting that this effect emerged despite the diminutive power allowed by the nine-participant dysphoric male group. Of possible relevance, Siegle and colleagues (2001) found that a predominantly male depressed sample showed faster reaction times to negative than positive nonpersonally relevant words. However, one might also argue that the marginal effect found in the current study could arise out of such a small sample due to an anomalous

confound. For example, the dysphoric male group reported greater incidence of parental depression than the dysphoric female group. Nonetheless, because the study's planned contrasts became modified by the significant contribution of gender, this exploration was thought to be warranted, and may, at the least, generate a question for future research to examine: Do dysphoric males, and not dysphoric females respond significantly faster to negative than to positive information on emotional information processing tasks?

Planned analyses also revealed a significant main effect of dysphoria status, as well as a qualifying dysphoria status by gender interaction effect. That is, on the whole, the dysphoric group responded more quickly than the nondysphoric group following emotional word presentations. But, this effect was driven by the strong tendency for dysphoric males to respond more quickly than nondysphoric males to emotional words. In fact, dysphoric females exhibited *slower* reaction times than nondysphoric females. When comparing males and females within the dysphoric group, dysphoric males showed faster reaction times than dysphoric females. However, when comparing males and females within the nondysphoric group, nondysphoric males showed *slower* reaction times than nondysphoric females. This last finding may be in accord with research that has suggested that women tend to be better processors of emotional information than men. This idea will be reviewed more later.

Relevant information processing and vulnerability research might help explain the reaction time results in the current study. To interpret results from an attentional

study using a dichotic listening paradigm, Ingram, Bernet, and McLaughlin (1994) posited that the early pre-attentive processing, which normally sifts self-relevant from non-self-relevant information, can become disproportionately more efficient (i.e., biased) at identifying emotional information than nonemotional information in depression vulnerable individuals. Similar results for increased sensitivity to emotional stimuli, independent of specific valence, have been found in the depression (e.g., Matthews & Southall, 1991) and in the anxiety disorders literature (e.g., Martin, Williams, & Clark, 1991; Mogg & Marden, 1990). It is possible that such an “emotional early warning system” (Ingram et al., 1994, p. 328) could explain the tendency for dysphoric males to identify emotional words more quickly than nondysphoric males and nondysphoric females. However, results within the female group were in direct contrast with those found in males. An attempt to superimpose the early warning system framework would suggest that nondysphoric females were more attuned to emotional information than nondysphoric males, but that dysphoric females were *less* attuned to emotional information. If becoming sensitized to emotional information over non-emotional information were an indicator of increased risk for depressotypic information processing, dysphoria in females would appear to *decrease* “maladaptive” early attentional processing. This conclusion is unsatisfactory.

More recently, Amir and colleagues (1996) found that socially phobic individuals’ interference effects from socially-threatening stimuli were attenuated under conditions of high anxiety. The authors suggested that increased effort may

have enabled individuals to inhibit intrusive emotionally-relevant information. In the present study it is possible that dysphoric females, who were experiencing moderate sadness and anxiety, could have exhibited a suppression effect. Emotional words, which could have been viewed as threatening to current emotional state, may have evoked greater global cognitive effort, resulting in brief interference of what might otherwise have been highly efficient processing. A review of the literature failed to unearth reports of main or interaction effects of gender on reaction time pertinent to emotional information processing. Because no nonemotional task was conducted in the current study, contrasts between emotional and nonemotional information processing within participants could not be made.

While motivated effort could affect reaction times, the possibility of indecision as an alternate explanation was also worth exploring. The dysphoric group endorsed greater indecisiveness than the nondysphoric group on the BDI, however dysphoric males and dysphoric females endorsed relatively equal levels of indecisiveness. Thus, there was no evidence to suggest that indecisiveness explained the gender by dysphoria status interaction reaction time findings. Lastly, dysphoric males in this sample endorsed a higher parental incidence of depression than the dysphoric females in the sample. It is possible that genetic contributions to depression vulnerability could be expressed or mediated through a predisposition to process information in depressotypic fashion when in a dysphoric mood.

Pupil Dilation Findings

It was predicted that dysphoric individuals would exhibit depressotypic biases as compared to nondysphoric individuals. Specifically, it was hypothesized that dysphoric individuals would exhibit greater sustained pupil dilation in response to negative words as compared to nondysphoric individuals. Main effects for gender, dysphoria status (i.e., sustained dilation irrespective of word valence), and valence were not predicted. These main effects, along with two- and three-way interactions outside the interaction of principle interest (e.g., dysphoria status x valence) among these three independent variables, were tested to rule out potential confounds. Dual analytic strategies (e.g., consecutive series of significant data points, ANOVA with PCA components as dependent variables) were employed in order to best manage the potential for type I and type II errors when accounting for variance in pupil dilation data. The PCA identified a series of six distinct components that comprised the 12-second pupil dilation time course following each stimulus presentation. Overall, variance tended to be greater later in the time course, as might be expected by the conceptual shift from more automatic information processing early on to more effortful (i.e., intentional) processing later. Hypothesized results were expected to occur in the final four components, reflecting identification, decision, and elaboration processes.

In the current study, strong support for a dysphoria status by valence effect was lacking. Effects in the predicted directions (e.g. dysphoric group greater dilation to negative than nondysphoric group, dysphoric group greater dilation to negative

than positive), though loosely trending, did not reach acceptable alpha levels. This result was consistent with Siegle and colleagues (2001) findings that depressed individuals did not show significantly greater dilation to negative than positive words, compared to nondepressed individuals. In contrast, current results are inconsistent with Siegle et al. (2003), which reported briefly (i.e., 4.8-6.1s post-stimulus) greater elaborative processing following negative compared to positive words in depressed, relative to nondepressed, individuals. In another relevant study, which compared never depressed to formerly depressed individuals, Steidtmann (2006) reported that formerly depressed individuals exhibited significantly greater dilation following negative words in roughly the same region of the waveform as occurred in the present study. However, the effect size in the present study was smaller, and the use of a more conservative three-way ANOVA did not allow this effect to reach significance. Also, unlike previous studies, which found that depressed individuals exhibited greater elaborative processing to emotional words than nondepressed individuals (e.g., Siegle et al., 2001; Siegle et al., 2003), the present study found no main effects of dysphoria status at any point in the time course.

A significant effect of valence was found late in the pupil dilation time course, where dilation was greater following negative words than positive words. But this main effect was qualified by a gender by valence effect. Siegle and colleagues (2001) reported a main effect of valence such that both depressed and nondepressed individuals showed greater pupil dilation following negative words than following positive words about 1-3 seconds post-reaction time. However, in the present study

the interaction between word valence and gender was more appropriate for interpretation. For example, males did *not* exhibit greater dilation following negative compared to positive words.

Unexpectedly, significant effects of gender were found in the third component (roughly 2-4s), such that females showed greater dilation than males when collapsing across valence levels. This represented greater peak reactivity (e.g., stimulus identification/decision and early motor responses) to emotionally valenced information in females. As noted, the sole effect of gender was qualified by significant gender by valence interactions corresponding to the third component. Specifically, females showed greater dilation than males following negative words. Also, females showed less sustained dilation to positive words than males in the fifth component, and females exhibited greater sustained dilation following negative compared to positive words in the last component. The latter result qualified the aforementioned valence main effect in the last component. A study by Porter, Hood, Troscianko, and Macrae (2006) reported gender effects on pupil dilation that may be of some relevance to the current data. Specifically, females, but not males, showed greater sustained pupil dilation when presented with direct-gaze facial stimuli (compared to non-direct gaze faces) in the 3-7 seconds following stimulus onsets. The authors speculated that females may allocate greater effort to processing socially relevant information. It is likewise possible that females allocate greater effort than males to processing negatively valenced emotionally relevant information. An abundance of research suggests that women are better able to recognize, interpret, and

structurally encode emotional information than men (Grunwald et al., 1999; Thayer, Rossy, Ruiz-Padial, & Johnsen, 2003). In another study that may also inform the present results, Thayer and colleagues (2003) reported that women exhibited greater attention to emotions than men, but that this alone did not mediate increased depressive symptoms. Although women reporting elevated depressive symptoms exhibited greater attention to emotions, these women *additionally* exhibited impaired anti-rumination emotional coping strategies. Thus, heightened attention to emotions does not appear to be depressogenic in and of itself.

If women tend to be more conversant with verbal expression of emotions, are more socially focused, and are more vigilant to various forms of threat (Brody & Hall, 1993; Goodwin & Gotlib, 2004), it might be expected that women would more deeply process emotional words, particularly negative words. In some circumstances it may be adaptive to quickly recognize and apply greater processing resources to information conveying an unsatisfactory state of affairs or an implicit threat, particularly if something can be done to remedy the situation. On the other hand, persistent elaborative processing, or rumination, about negative information can be psychologically maladaptive. Relevant to this, significant dysphoria status by gender interactions were found very early and late in the time course. Immediately following stimulus presentations, dysphoric females showed greater dilation than nondysphoric females, whereas dysphoric males exhibited marginally *less* dilation than nondysphoric males. Interestingly, dysphoric females showed the greatest pupil dilation and nondysphoric females showed the least dilation among the four groups

during the initial component. Thus, it may be that in nondysphoric women immediate pre-attentive processing of emotional information is highly efficient, with minimal interference, which might explain the smaller cognitive load indicated by pupil dilation. If, as in Thayer et al. (2003), dysphoric females in this study were exhibiting impaired anti-rumination strategies, this could have led to early perceptual interference. For instance, ruminating about task-irrelevant problems could have led to lesser attentional engagement with the task.

With regard to males, late in the time course nondysphoric males showed greater pupil dilation than dysphoric males following emotional words. This result was largely explained by the fact that nondysphoric males showed significantly greater late dilation to positive words than dysphoric males. Dysphoric males constricted (i.e., dilation less than baseline dilation) at this point, similar to nondysphoric females and dysphoric females. Past research suggests that individuals' pupils might constrict if they find information to be boring, non-salient, or perhaps aversive (Chaney, Givens, Aoki, & Gombiner, 1989; Janisse, 1973; Trepagnier et al., 2006). In contrast, nondysphoric males' greater late dilation to positive words could represent a positivistic bias toward greater elaboration. With such a bias, the perception of positive words could lead to greater associative processing, which would presumably promote euthymic moods in a state sense (i.e., activation of existing positive associative networks) and trait sense (strengthening connections within positive associative networks). Of possible relevance, following positive word presentations *depressed* individuals have been observed to exhibit decreased levels of

sustained activity in the amygdala (Siegle, Steinhauer, Thase, Stenger, & Carter, 2002), a brain structure integrally linked to emotion processing. From a continuity perspective of depression, it would make intuitive sense that dysphoric males and females would not possess a positivistic elaborative bias.

Exploratory analyses using Guthrie and Buchwald's technique revealed several additional findings of interest. Dysphoric females showed greater dilation compared to nondysphoric females immediately following the presentation of positive words (e.g., sixth component). This effect could be due to increased interference when dysphoric females initially encounter emotionally dissonant information. In addition, a marginal effect was found later in the time course, with dysphoric females showing greater dilation following negative words than nondysphoric females. Joormann (2004) suggested that in the context of emotional information processing, dysphoria may be characterized by an inhibitory deficit for negative information. Although dysphoric females' reaction times to negative words were not facilitated, it is presumably possible that their sustained elaborative processing of negative information came about due to inefficient inhibition and degradation of negative cognitive content. Dysphoric females and dysphoric males did not significantly differ in sustained pupil dilation following words of any valence.

Given that females showed greater dilation to negative words than positive words, and that dysphoric females showed marginally greater dilation than dysphoric males to emotional words in general, it might have been expected that dysphoric

females would show greater dilation than dysphoric males following negative words. However, a significant difference was not found.

Within the nondysphoric portion of the sample, females showed considerably greater sustained dilation than males following negative words from 2-4 seconds following the stimulus onset, which occurs proximal to a participant's decision and task response. This result was consistent with the female emotional information processing advantage explanation posited earlier (i.e., deeper processing recruits greater cognitive resources). Late in the time course following positive words nondysphoric males showed greater sustained pupil dilation than nondysphoric females. Nondysphoric males continued to dilate nearly until the next stimulus presentation, while nondysphoric females constricted substantially below their baseline pupil diameter. Nondysphoric males' late sustained dilation following positive words distinguished them from dysphoric males, dysphoric females and nondysphoric females. Thus, the previously mentioned positivistic bias interpretation only applied to the nondysphoric male group.

Although relationships between participants' parental risk for depression and biases in sustained pupil dilation to valenced words were examined, findings suggested that parental risk status did not meaningfully contribute to differences in pupil dilation following positive or negative words.

Integrated Interpretation

Although reaction time, pupil dilation, and self-report measures may individually resemble the proverbial blind men examining seemingly unrelated sub-

structures of an elephant, simultaneous consideration of the three forms of data may facilitate explanations that can encompass and integrate macro- and micro-level phenomena related to emotional processing. Because gender by dysphoria status interaction effects were found, each of the four groups will be reviewed.

Dysphoric males

Dysphoric males exhibited less immediate cognitive load following emotional stimulus presentations and more quickly identified emotional words than nondysphoric males. This pattern could reflect a facilitative effect of dysphoria on the processing of lexical emotional content in males. That is, persistent dysphoric mood might lead to more efficient emotional processing, which would reduce immediate cognitive load. Dysphoric males also showed considerably diminished elaborative processing late in the time course following positive words. Given that this pupil constriction pattern was also seen in the waveforms of dysphoric females but not nondysphoric males, one might interpret this as a lack of a positivistic late elaborative bias.

Dysphoric females

Dysphoric females showed delayed reaction times and relatively increased pupil dilation immediately following the presentation of emotional words, particularly positive words. It is possible that this reflected an immediate interference effect, where greater conflict arose in pre-attentive cognitive processes. Later in the waveform, dysphoric females did not exhibit greater processing following positive words, but did show marginally greater cognitive load than nondysphoric females

following negative words. This specific pattern in dysphoric females suggests that despite a generally increased pre-attentive cognitive load for all emotional words, lesser inhibition, greater sustained activation, or a combination of the two processes related to specifically negative words occurred, leading to greater late elaboration following negative words. In a possibly consistent finding, dysphoric females endorsed significantly greater rumination tendencies than nondysphoric females. Thus, it is possible that the marginally greater late elaboration following negative words shown by dysphoric females' could represent subtle proto-ruminative cognitive processes, similar to those suggested to exist in depressed individuals (Siegle et al., 2003).

Nondysphoric females

In the current study nondysphoric females were expected to represent a low-risk group. Nondysphoric females rapidly identified the emotions of words and showed relatively low immediate dilation. As suggested earlier, this might reflect efficient processing of lexical emotional content with minimal interference. As noted earlier, extensive research supports the idea that greater emotional attunement might be the *modus operandi* of psychologically healthy females, and need not represent an indicator of increased cognitive vulnerability. Thus, nondysphoric females' lesser immediate dilation to positive words, when compared to dysphoric females, could reflect this efficiency and lack of interference.

Within the timeframe immediately preceding and following the decision and response (e.g., peak dilation) to negative words, nondysphoric females exhibited pupil

dilation roughly equivalent to dysphoric females, which represented greater cognitive load than that of nondysphoric males. This suggested that in women these cognitive processes were unaffected by the presence of dysphoria. While an efficiency explanation might predict lesser cognitive peak load for nondysphoric females, research suggesting that lexical emotional stimuli are experienced as more intense by females (Grunwald et al., 1999) might lead one to accept a threat-evaluation and arousal explanation. That is, greater cognitive resources may have been usurped by threat evaluation, greater arousal, and deeper associative processes within more complex cognitive-emotional structures, leading to higher peak dilation in nondysphoric (and dysphoric) women than in nondysphoric men. In addition, nondysphoric females showed *lesser* late cognitive load than nondysphoric males following positive words. A plausible explanation for this finding was not produced from a search of relevant literature.

Nondysphoric males

Nondysphoric males exhibited slowed reaction times and marginally increased pupil dilation immediately following emotional word presentations, which could represent relative processing inefficiency. However, nondysphoric males exhibited less peak dilation than dysphoric males and nondysphoric females following negative words. It would be possible for cognitive load to be less at this valence identification and response stage of processing if nondysphoric males experienced the words as less intensely arousing or salient, as compared to dysphoric males and nondysphoric females. Late in the time course, nondysphoric males exhibited considerably greater

cognitive load following positive words as compared to negative words. As suggested previously, nondysphoric males' late sustained pupil dilation following positive words might constitute a bias for elaborating incoming positive information.

Vulnerability to Depression

At the time of the experiment, none of the participants endorsed a current or past syndrome consistent with a major depressive episode. However, of the four groups, nondysphoric males appeared to be least susceptible to developing depression according to information processing patterns shown in the current data. This would seem consistent with epidemiological and gender research on depression, which has consistently shown an approximate prevalence ratio of 2:1 between women and men. Although nondysphoric females' more efficient processing of emotional information may not in and of itself indicate greater vulnerability, when combined with a greater tendency to ruminate, significant life stress could trigger a positive feedback circuit between attention to emotions and depressive rumination. Nondysphoric women in this sample endorsed a greater tendency to ruminate, and therefore, it is possible that they possess incrementally greater risk than nondysphoric men in the study. All else being equal, it might have been expected that dysphoric females would show the same or increasingly facilitated initial processing of emotional words, with shorter reaction times and lesser immediate dilation than nondysphoric females. Interestingly, opposite findings of slower reaction time and greater immediate dilation in dysphoric females may be evidence for an initial interference effect that briefly inhibits efficient

processing of incoming emotional information. Results indicate that in the absence of subsequent distracting information streams, this blunting effect was short-lived following negative words, as dysphoric females showed increased cognitive load later in the time course. Given that this group endorsed a frequent tendency to ruminate, it is likely that they would be at significant risk of experiencing a depressive episode in the future. Similarly dysphoric males in the study endorsed elevated levels of rumination and exhibited information processing patterns in VID task performance that might make these individuals more vulnerable to future depression. Dysphoric males exhibited facilitated processing of emotional information and appeared to lack the positivistic elaborative processing bias that was present in nondysphoric males.

Limitations

The current study had several limitations. Although the investigation was interested in examining phenomena related to vulnerability to depression, its cross-sectional design only allowed speculative interpretations of aspects that distinguished groups thought to be at greater risk of developing depression. Only longitudinal follow up could have verified whether these dysphoric individuals were more likely to become depressed and whether reported differences in behavioral, physiological, or self-report measures could predict this. Also, while vulnerability to depression was a primary interest of the study, the current dysphoric sample was also characterized by significantly elevated anxiety. It is possible that the combined elevations of anxiety and depression within the dysphoric group increased heterogeneity in the group's reaction time and physiological data, as anxiety and depression are typically

associated with different cognitive responses to specific types of emotional stimuli (e.g., Bradley, Mogg, Millar, & White, 1995; Dalgleish et al., 2003; Mogg, Bradley, Williams, & Mathews, 1993). This would have decreased the likelihood of detecting differences between the dysphoric and nondysphoric groups. Therefore, current results may not generalize to dysphoric populations characterized predominantly by symptoms of depression with minimal anxiety. Nonetheless, these results may be relevant to a common subset within clinical populations that present with comorbid symptoms of depression and anxiety. Additionally, because interaction effects related to gender had not been expected, the present study may have been under-powered to detect subtle effects. It is possible that some of the medium effects with p-values in the .10-.15 range would have fallen below $\alpha = .05$ with increased power from having at least 20 participants in each of the three-way interaction cells. With this shortcoming in mind the current study has reported and, to some extent, made cautious interpretation of statistical trends where effect sizes have suggested it might be warranted. This less-conservative approach was also adopted due to the exploratory nature of the study and its mission to identify areas in need of future investigation. Thus, results from this study need to be replicated by future research.

In addition, at the present time, interpreting the meaning of pupil dilation, while tempting, can be precarious. Evidence has accumulated to support the notion that measuring pupil dilation reliably indexes cognitive load. However, once greater load is identified, investigators are far less certain of what kind of processing is occurring. For example, Dionisio, Granholm, Hillix, and Perrine (2001) found that

individuals exhibited greater pupil dilation when asked to confabulate a deceptive rather than a true answer. It has also been proposed that proto-ruminative thoughts can lead to greater dilation (Siegle et al., 2003). Thus, it is possible that intentionally directed thoughts, as well as unintentional intrusive thoughts can contribute to greater cognitive load, and thereby greater pupil dilation. This presents interpretation problems within the contexts of dysphoria and depression. For example, it is possible for two individuals show remarkably similar elevated dilation waveforms following a negative word, with one experiencing a sequence of intrusive negative associations to the word, while the other person intentionally focuses on a positive, distracting daydream. One person might be adept at self-regulating emotions, while the other might be in need of clinical intervention. Better methods of assessing thoughts either during or immediately following tasks could better inform which kinds of thoughts may be contributing to greater load and dilation in particular groups of interest.

Implications

Despite these limitations, the present study highlighted areas in need of further research, and its findings may have important implications. Because participants endorsed either a stable dysphoric or nondysphoric mood, findings may be clinically and theoretically relevant. Furthermore, because history of depression was ruled out in dysphoric participants, findings may have relevance to processes that occur in individuals prior to a first onset of depression. To the investigator's knowledge this is the first study to utilize reaction time and pupil dilation measures to examine the performance of never depressed, dysphoric individuals on an emotional information

processing task. Future investigations should follow up to confirm and elucidate seemingly complex relationships among dysphoria, gender, and emotional information processing. The present findings also suggest the value of a future investigation that could contrast emotional *and* nonemotional information processing in dysphoric individuals as Siegle and colleagues' (2001) did with depressed individuals.

In a broader context, findings from the present study are generally consistent with cognitive theories of depression. Dysphoria has been identified as a subclinical syndrome that places individuals at greater risk for depression, and consistent with continuity theories of depression, dysphoria was associated with depressotypic biases in attentional processing of emotional information in this study. Cognitive theories of depression (e.g., Beck, 1967; Ingram, 1984; Teasdale, 1983) have largely focused on how selective attention might be biased toward negative information in depressed and depression vulnerable individuals, and this appeared to occur in dysphoric females in this study. However, certain findings from this study and others (e.g., Gotlib, McLachlan, & Katz, 1988; Karparova, Kersting, & Suslow, 2007; Koster, De Raedt, Goeleven, Franck, & Crombez, 2005) suggest that deficient attentional processing of *positive* information might also be related to the development and maintenance of depression. If sufficient supporting evidence accumulates, cognitive theories of depression may need to explain processes that could lead to the elimination of a positivistic bias in depression vulnerable individuals. Lastly, the present study

provides further support for the use of pupillometry in studying vulnerability to depression.

Conclusion

The current investigation produced several important findings. Although results suggested that dysphoria status does not bias individuals' information processing in a uniform way, there was some evidence that dysphoria, through an interaction with gender, may produce patterns of information processing that appear depressotypic, or possibly even depressogenic. On the other hand, observed differences in information processing could merely be correlates of stable dysphoria. Gender and dysphoria status interactions were prominent in both behavioral and psychophysiological data. For males the presence of dysphoria had a facilitative effect on early processing of emotional information, whereas for females early processing was temporarily impeded. Both dysphoric males and females showed evidence of depressotypic late processing, but in regard to different valenced information. Males showed diminished processing following positive words, and females showed marginally greater processing following negative words. Ultimately, findings from this study highlight the importance of examining relationships between gender and cognition when comparing the emotional information processing of populations deemed to be vulnerable to depression. If these results are replicable, they may identify a gap or a need for synthesis among the information processing, vulnerability, and psychophysiology literatures to explain the observed relationships among gender, dysphoria, and emotionally valenced information.

Footnotes

1. It is possible that participants who scored in the same dysphoria status range during the prescreen and experimental session could have, at some point in the intervening time period, fallen out of that range. Although the present design cannot eliminate this possibility, it is difficult to see how this could have done anything other than diminish any effects of dysphoria.
2. Although BDI item #13 assesses global difficulties in decision making, the modal score for the dysphoric group was “I put off making decisions more than I used to,” suggesting a motivational quality to the decision making difficulties. Nonetheless, six dysphoric participants endorsed “I have greater difficulty in making decisions than before,” which may be more consistent with neurocognitive deficits commonly observed in depressive syndromes. Thus, while the analysis of this item imperfectly relates to decision making processes embedded in reaction times, it may be worthy of qualified consideration.
3. One dysphoric male participant did not complete the Family History Screen.
4. A five-component model (ala Siegle et al., 2001) was forced onto the data, however one of the components was rendered un-interpretable. Other exploratory models were tested, however each of these inadequately characterized the structure of the data.

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Appendix A

Beck Depression Inventory

On this questionnaire are groups of statements. Please read each group of statements carefully. Then pick out the one statement that best describes the way you have been feeling the PAST TWO WEEKS, INCLUDING TODAY! Circle the number beside the statement you picked. If several statements in the group seem to apply equally well, circle each one. Be sure to read all the statements in each group before making your choice.

1. 0 I do not feel sad.
1 I feel sad.
2 I am sad all the time and I can't snap out of it.
3 I am so sad or unhappy that I can't stand it.
2. 0 I am not particularly discouraged about the future.
1 I feel discouraged about the future.
2 I feel I have nothing to look forward to.
3 I feel that the future is hopeless and that things cannot improve.
3. 0 I do not feel like a failure.
1 I feel I have failed more than the average person.
2 As I look back on my life, all I can see is a lot of failures.
3 I feel I am a complete failure as a person.
4. 0 I get as much satisfaction out of things as I used to.
1 I don't enjoy things the way I used to.
2 I don't get real satisfaction out of anything anymore.
3 I am dissatisfied or bored with everything.
5. 0 I don't feel particularly guilty.
1 I feel guilty a good part of the time.
2 I feel quite guilty most of the time.
3 I feel guilty all of the time.
6. 0 I don't feel disappointed in myself.
1 I am disappointed in myself.
2 I am disgusted with myself.
3 I hate myself.
7. 0 I don't feel I am being punished.
1 I feel I may be punished.
2 I expect to be punished.
3 I feel I am being punished.
8. 0 I don't feel I am any worse than anybody else.
1 I am critical of myself for my weakness or mistakes.
2 I blame myself all of the time for my faults.
3 I blame myself for everything bad that happens.
9. 0 I don't have thoughts of killing myself.
1 I have thoughts of killing myself, but I would not carry them out.
2 I would like to kill myself.
3 I would kill myself if I had the chance.

10. 0 I don't cry any more than usual.
 1 I cry more now than I used to.
 2 I cry all the time now.
 3 I use to be able to cry, but now I can't cry though I want to.
11. 0 I am no more irritated now than I ever am.
 1 I get annoyed or irritated more easily than I used to.
 2 I feel irritated all the time now.
 3 I don't get irritated at all by the things that used to irritate me.
12. 0 I have not lost interest in other people.
 1 I am less interested in other people than I used to be.
 2 I have lost most of my interest in other people.
 3 I have lost all of my interest in other people.
13. 0 I make decisions about as well as I ever could.
 1 I put off making decisions more than I used to.
 2 I have greater difficulty in making decisions than before.
 3 I can't make decisions at all anymore.
14. 0 I don't feel I look any worse than I used to.
 1 I am worried that I am looking old or unattractive.
 2 I feel that there are permanent changes in my appearance that make me look unattractive.
 3 I believe that I look ugly.
15. 0 I can work about as well as before.
 1 It takes an extra effort to get started at something.
 2 I have to push myself very hard to do anything.
 3 I can't do any work at all.
16. 0 I can sleep as well as usual.
 1 I don't sleep as well as I used to.
 2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep.
 3 I wake up several hours earlier than I used to and cannot get back to sleep.
17. 0 I don't get more tired than usual.
 1 I get tired more easily than I used to.
 2 I get tired from doing almost anything.
 3 I am too tired to do anything.
18. 0 My appetite is no worse than usual.
 1 My appetite is not as good as it used to be.
 2 My appetite is much worse now.
 3 I have no appetite at all anymore.
19. 0 I haven't lost much weight, if any, lately.
 1 I have lost more than 5 pounds. I am purposely trying to lose weight.
 2 I have lost more than 10 pounds. By eating less? Yes _____ No _____.
 3 I have lost more than 15 pounds.

20. 0 I am no more worried about my health than usual.
1 I am worried about physical problems such as aches and pains; or upset stomach; or constipation.
2 I am very worried about physical problems and it's hard to think of much else.
3 I am so worried about my physical problems that I cannot think of anything else.
21. 0 I have not noticed any recent changes in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

Appendix B

Beck Anxiety Inventory (BAI)

A list of common symptoms of anxiety will be presented. Indicate how much you have been bothered by each symptom during the PAST WEEK, INCLUDING TODAY by choosing the number of the corresponding description beneath the symptom.

	1	2	3	4
	Not at all	Mildly:	Moderately: It did not bother me	Severely: It was very unpleasant, but I could stand it
1. Numbness or tingling	1	2	3	4
2. Feeling hot	1	2	3	4
3. Wobbliness in legs	1	2	3	4
4. Unable to relax	1	2	3	4
5. Fear of the worst happening	1	2	3	4
6. Dizzy or lightheaded	1	2	3	4
7. Heart pounding or racing	1	2	3	4
8. Unsteady	1	2	3	4
9. Terrified	1	2	3	4
10. Nervous	1	2	3	4
11. Feeling of choking	1	2	3	4
12. Hands trembling	1	2	3	4
13. Shaky	1	2	3	4
14. Fear of losing control	1	2	3	4
15. Difficulty breathing	1	2	3	4
16. Fear of dying	1	2	3	4
17. Scared	1	2	3	4
18. Indigestion or discomfort in abdomen	1	2	3	4
19. Faint	1	2	3	4
20. Face flushed	1	2	3	4
21. Sweating (not due to heat)	1	2	3	4

Appendix C

INVENTORY TO DIAGNOSE DEPRESSION – LIFETIME VERSION

Try to remember THE WEEK IN YOUR LIFE YOU FELT THE MOST DEPRESSED.

What was the approximate starting and ending date of the episode you have in mind?
began: _____ ended: _____

Circle the number of the one statement that best describes how you felt. Remember to also circle whether you felt that way for MORE or LESS than two weeks.

- 1) 0 I did not feel sad or depressed.
1 I occasionally felt sad or down.
2 I felt sad most of the time, but I was able to snap out of it.
3 I felt sad all the time, and I couldn't snap out of it.
4 I was so sad or unhappy that I couldn't stand it.

This lasted MORE/LESS than two weeks (circle one)

- 2) 0 My energy level was normal.
1 My energy level was a little lower than normal.
2 I got tired more easily and had less energy than is usual.
3 I got tired from doing almost anything.
4 I felt tired or exhausted almost all the time.

This lasted MORE/LESS than two weeks (circle one)

- 3) 0 I was not feeling more restless and fidgety than usual.
1 I felt a little more restless or fidgety than usual.
2 I was very fidgety, and I had some difficulty sitting still in a chair.
3 I was extremely fidgety, and I paced a little bit almost everyday.
4 I paced more than an hour per day, and I couldn't sit still.

This lasted MORE/LESS than two weeks (circle one)

- 4) 0 I did not talk or move more slowly than usual.
1 I talked a little slower than usual.
2 I spoke slower than usual, and it took me longer to respond to questions, but I could still carry on a normal conversation.
3 Normal conversations were difficult for me because it was hard to start talking.
4 I felt extremely slowed down physically, like I was stuck in mud.

This lasted MORE/LESS than two weeks (circle one)

- 5) 0 I did not lose interest in my usual activities.
1 I was a little less interested in 1 or 2 of my usual activities.
2 I was less interested in several of my usual activities.
3 I lost most of my interest in almost all of my usual activities.
4 I lost interest in all of my usual activities.

This lasted MORE/LESS than two weeks (circle one)

- 6) 0 I got as much pleasure out of my usual activities as usual.
1 I got a little less pleasure from 1 or 2 of my usual activities.
2 I got less pleasure from several of my usual activities.
3 I got almost no pleasure from several of my usual activities.
4 I got no pleasure from any of the activities which I usually enjoy.

This lasted MORE/LESS than two weeks (circle one)

- 7) 0 My interest in sex was normal.
1 I was only slightly less interested in sex than usual.
2 There was a noticeable decrease in any interest in sex.
3 I was much less interested in sex then.
4 I lost all interest in sex.

This lasted MORE/LESS than two weeks (circle one)

- 8) 0 I did not feel guilty.
1 I occasionally felt a little guilty.
2 I often felt guilty.
3 I felt quite guilty most of the time.
4 I felt extremely guilty most of the time.

This lasted MORE/LESS than two weeks (circle one)

- 9) 0 I did not feel like a failure.
1 My opinion of myself was occasionally a little low.
2 I felt I was inferior to most people.
3 I felt like a failure.
4 I felt I was a totally worthless person.

This lasted MORE/LESS than two weeks (circle one)

- 10) 0 I didn't have any thoughts of death or suicide.
1 I occasionally thought life was not worth living.
2 I frequently thought of dying in passive ways (such as going to sleep and not waking up) or that I'd be better off dead.
3 I had frequently thoughts of killing myself.
4 I tried to kill myself.

This lasted MORE/LESS than two weeks (circle one)

- 11) 0 I could concentrate as well as usual.
1 My ability to concentrate was slightly worse than usual.
2 My attention span was not as good as usual and I had difficulty collecting my thoughts; but this didn't cause any problems.
3 My ability to read or hold a conversation was not as good as usual.
4 I could not read, watch TV, or have a conversation without great difficulty.

This lasted MORE/LESS than two weeks (circle one)

- 12) 0 I made decisions as well as usual.
1 Decision making was slightly more difficult than usual.
2 It was harder and took longer to make decisions, but I did make them.
3 I was unable to make some decisions.
4 I couldn't make any decisions at all.

This lasted MORE/LESS than two weeks (circle one)

- 13) 0 My appetite was not less than normal.
1 My appetite was slightly worse than usual.
2 My appetite was clearly not as good as usual, but I still ate.
3 My appetite was much worse.
4 I had no appetite at all, and I had to force myself to eat even a little.

This lasted MORE/LESS than two weeks (circle one)

- 14) 0 I didn't lose any weight.
1 I lost less than 5 pounds.
2 I lost between 5-10 pounds.
3 I lost between 11-25 pounds.
4 I lost more than 25 pounds.

This lasted MORE/LESS than two weeks (circle one)

- 15) 0 My appetite was not greater than normal.
1 My appetite was slightly greater than usual.
2 My appetite was clearly greater than usual.
3 My appetite was much greater than usual.
4 I felt hungry all the time.

This lasted MORE/LESS than two weeks (circle one)

- 16) 0 I didn't gain any weight.
1 I gained less than 5 pounds.
2 I gained between 5-10 pounds.
3 I gained between 11-25 pounds.
4 I gained more than 25 pounds.

This lasted MORE/LESS than two weeks (circle one)

- 17) 0 I was not sleeping less than usual.
1 I occasionally had light difficulty sleeping.
2 I clearly didn't sleep as well as usual.
3 I slept about half my normal amount of time.
4 I slept less than 2 hours per night.

This lasted MORE/LESS than two weeks (circle one)

- 18) 0 I was not sleeping more than normal.
1 I occasionally slept more than usual.
2 I frequently slept at least 1 hour more than usual.
3 I frequently slept at least 2 hours more than usual.
4 I frequently slept at least 3 hours more than usual.

This lasted MORE/LESS than two weeks (circle one)

- 19) 0 I did not feel anxious, nervous, or tense.
1 I occasionally felt a little anxious.
2 I often felt anxious.
3 I felt anxious most of the time.
4 I felt terrified and near panic.

This lasted MORE/LESS than two weeks (circle one)

- 20) 0 I did not feel discouraged about the future.
1 I occasionally felt a little discouraged about the future.
2 I often felt discouraged about the future.
3 I felt very discouraged about the future most of the time.
4 I felt that the future was hopeless and that things would never improve.

This lasted MORE/LESS than two weeks (circle one)

- 21) 0 I did not feel irritated or annoyed.
1 I occasionally got a little more irritated than usual.
2 I got irritated or annoyed by things that usually didn't bother me.
3 I felt irritated or annoyed almost all the time.
4 I felt so depressed that I didn't get irritated at all by things that would normally bother me.

This lasted MORE/LESS than two weeks (circle one)

- 22) 0 I was not worried about my physical health.
1 I was occasionally concerned about bodily aches and pains.
2 I was worried about my physical health.
3 I was very worried about my physical health.
4 I was so worried about my physical health that I could not think about anything else.

This lasted MORE/LESS than two weeks (circle one)

- 23) 0 This bout of depression is the only one I have ever had.
1 I have had an additional period of depression similar to the one I already described.
2 I have had two more periods of depression similar to the one I already described.
3 I have had three more periods of depression similar to the one I already described.
4 I have had four or more periods of depression similar to the one I already described.

- 24) 0 I did not get any treatment for how I felt.
1 I got psychotherapy, but did not take anti-depressant medication.
2 I took anti-depressant medication, but did not get psychotherapy.
3 I got psychotherapy and took anti-depressant medication(s).
4 I was admitted to a psychiatric hospital for treatment.

Appendix D

Positive and Negative Affect Schedule (PANAS)

This scale consists of a number of words that describe different feelings and emotions. Read

each item and then mark the appropriate answer in the space next to that word.

Indicate to what extent you feel this way right now, that is, at the present moment.

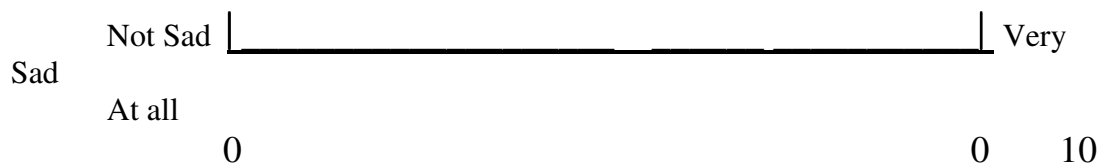
Use the following scale to record your answers:

1	2	3	4	5
very slightly or not at all	a little	moderately	quite a bit	extremely
___ interested			___ irritable	
___ distressed			___ alert	
___ excited			___ ashamed	
___ upset			___ inspired	
___ strong			___ nervous	
___ guilty			___ determined	
___ scared			___ attentive	
___ hostile			___ jittery	
___ enthusiastic			___ active	
___ proud			___ afraid	

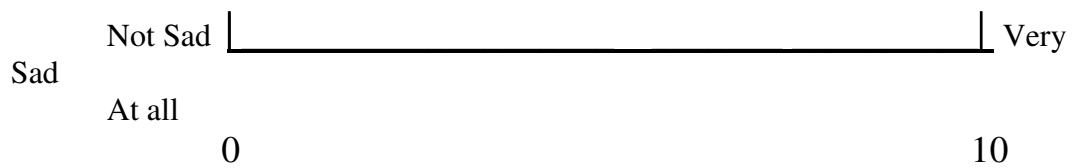
Appendix E

Mark an χ on the line which indicates a range of feelings. For instance, if you were feeling “moderate sadness” you would indicate on the line below as such:

Example:



Please make your mark here:



Appendix F

Structured Clinical Interview for DSM-IV

SCID-I (DSM-IV) Version 2.0

Past MDE (FEB 1996 FINAL)

Mood Episodes A. 12

PAST MAJOR DEPRESSIVE EPISODE*

MDE CRITERIA

-> IF NOT CURRENTLY DEPRESSED:
Have you ever had a period
when you were feeling de-
pressed or down most of the
day nearly every day? (What
was that like?)

-> IF CURRENTLY DEPRESSED BUT
FULL CRITERIA ARE NOT MET,
SCREEN FOR PAST MDE: Has
there ever been another time
when you were depressed or
down most of the day nearly
every day? (What was that
like?)

IF YES: When was that? How
long did it last? (As long
as two weeks?)

-> IF PAST DEPRESSED MOOD:
During that time, did you
lose interest or pleasure
in things you usually enjoyed?
(What was that like?)

-> IF NO PAST DEPRESSED MOOD:
What about a time when you
lost interest or pleasure
in things you usually enjoyed?
(What was that like?)

IF YES: When was that? Was
it nearly every day? How
long did it last? (As long as
two weeks?)

Have you had more than one time
like that? (Which time was the
worst?)

IF UNCLEAR: Have you had any
times like that in the past year?

A. Five or more of the fol-
lowing symptoms have been
present during the same two-
week period and represent a
change from previous func-
tioning; at least one of
the symptoms was either
(1) depressed mood or
(2) loss of interest or
pleasure.

(1) depressed mood most of
the day, nearly every day,
as indicated by either sub-
jective report (e.g., feels
sad or empty) or observation
made by others (e.g., appears
tearful). Note: in children
and adolescents, can be
irritable mood.

(2) markedly diminished
interest or pleasure in all,
or almost all, activities
most of the day, nearly
every day (as indicated
either by subjective
account or observation
made by others)

? 1 2 3

A52

? 1 2 3

A53

IF NEITHER
ITEM (1)
NOR (2) IS
CODED "3,"
GO TO
*CURRENT
MANIC EPI-
ISODE,*
A. 18

NOTE: IF MORE THAN ONE PAST
EPISODE IS LIKELY, SELECT THE
"WORST" ONE FOR YOUR INQUIRY
ABOUT A PAST MAJOR DEPRESSIVE
EPISODE. HOWEVER, IF THERE WAS
AN EPISODE IN THE PAST YEAR, ASK
ABOUT THAT EPISODE EVEN IF IT
WAS NOT THE WORST.

?=inadequate information

1=absent or false

2=subthreshold

3=threshold or true

FOR THE FOLLOWING QUESTIONS,
FOCUS ON THE WORST TWO WEEKS
OF THE PAST MAJOR DEPRESSIVE
EPISODE THAT YOU ARE INQUIRING
ABOUT

During that (TWO WEEK PERIOD)...

..did you lose or gain any
weight? (How much?) (Were you
trying to lose weight?)

IF NO: How was your appe-
tite? (What about compared
to your usual appetite?)
(Did you have to force your-
self to eat?) (Eat [less/
more] than usual?) (Was that
nearly every day?)

NOTE: WHEN RATING THE FOLLOWING ITEMS,
CODE "1" IF CLEARLY DIRECTLY DUE TO A
GENERAL MEDICAL CONDITION, OR TO MOOD-
INCONGRUENT DELUSIONS OR HALLUCINATIONS

(3) significant weight loss ? 1 2 3
when not dieting, or weight
gain (e.g., a change of more
than 5% of body weight in a
month), or decrease or increase
in appetite nearly every day.
Note: in children, consider
failure to make expected
weight gains.

Check if:

___ weight loss or decreased
appetite
___ weight gain or increased
appetite

A54

A55

A56

..how were you sleeping?
(Trouble falling asleep, waking
frequently, trouble staying
asleep, waking too early, OR
sleeping too much? How many
hours a night compared to usual?
Was that nearly every night?)

(4) insomnia or hypersomnia ? 1 2 3
nearly every day

Check if:

___ insomnia
___ hypersomnia

A57

A58

A59

..were you so fidgety or rest-
less that you were unable to
sit still? (Was it so bad that
other people noticed it? What did
they notice? Was that nearly
every day?)

(5) psychomotor agita-
tion or retardation ? 1 2 3
nearly every day (observ-
able by others, not merely
subjective feelings of
restlessness or being
slowed down)

Check if:

___ psychomotor agitation
___ psychomotor retardation

A60

A61

A62

IF NO: What about the op-
posite -- talking or mov-
ing more slowly than is
normal for you? (Was it so
bad that other people
noticed it? What did they
notice? Was it nearly every day?)

..what was your energy like?
(Tired all the time? Nearly
every day?)

(6) fatigue or loss of
energy nearly every day ? 1 2 3

A63

?=inadequate information 1=absent or false 2=subthreshold 3=threshold or true

During that time...

..how did you feel about yourself? (Worthless?)
(Nearly every day?)

IF NO: What about feeling guilty about things you had done or not done? (Nearly every day?)

(7) Feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick)

? 1 2 3

A64

NOTE: CODE "1" OR "2" FOR LOW SELF-ESTEEM BUT NOT WORTHLESSNESS

Check if:

___ worthlessness
___ inappropriate guilt

A65

A66

..did you have trouble thinking or concentrating? (What kinds of things did it interfere with?)
(Nearly every day?)

IF NO: Was it hard to make decisions about everyday things? (Nearly every day?)

(8) diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others)

? 1 2 3

A67

Check if:

___ diminished ability to think
___ indecisiveness

A68

A69

..were things so bad that you were thinking a lot about death or that you would be better off dead? What about thinking of hurting yourself?

IF YES: Did you do anything to hurt yourself?

(9) recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide

? 1 2 3

A70

NOTE: CODE "1" FOR SELF-MUTILATION W/O SUICIDAL INTENT

Check if:

___ thoughts of own death
___ suicidal ideation
___ specific plan
___ suicide attempt

A71

A72

A73

A74

?=inadequate information 1=absent or false 2=subthreshold 3=threshold or true

AT LEAST FIVE OF THE ABOVE SXS
[A(1-9)] ARE CODED "3" AND AT LEAST
ONE OF THESE IS ITEM (1) OR (2)

1

3

A75

CONTINUE
BELOW

IF NOT ALREADY ASKED: Has
there been any other time when
you were (depressed/OWN
WORDS) and had even more of the
symptoms than I just asked you
about?

-> IF YES: RETURN TO *PAST MAJOR
DEPRESSIVE EPISODE,* A. 12, AND
CHECK WHETHER THERE HAVE BEEN
ANY OTHER MAJOR DEPRESSIVE
EPISODES THAT WERE MORE SEVERE
AND/OR CAUSED MORE SYMPTOMS. IF
SO, ASK ABOUT THAT EPISODE.

-> IF NO: GO TO *CURRENT MANIC
EPISODE,* A. 18.

NOTE: DSM-IV criterion B (i.e.,
does not meet criteria for a
Mixed Episode) has been omitted
from the SCID.

IF UNCLEAR: Has (depres-
sive episode/OWN WORDS)
made it hard for you to do
your work, take care of things
at home, or get along with
other people?

C. The symptoms cause clinic-
ally significant distress or
impairment in social, occu-
pational, or other important
areas of functioning.

?

1

2

3

A76

CONTINUE
ON NEXT
PAGE

IF NOT ALREADY ASKED: Has
there been any other time when
you were (depressed/OWN
WORDS) and it caused even more
problems than the time I just
asked you about?

-> IF YES: RETURN TO *PAST MAJOR
DEPRESSIVE EPISODE,* A. 12, AND
CHECK WHETHER THERE HAVE BEEN
ANY OTHER MAJOR DEPRESSIVE
EPISODES THAT WERE MORE SEVERE
AND/OR CAUSED MORE SYMPTOMS. IF
SO, ASK ABOUT THAT EPISODE.

-> IF NO: GO TO *CURRENT MANIC
EPISODE,* A. 18.

?=inadequate information 1=absent or false 2=subthreshold 3=threshold or true

Just before this began, were you physically ill?

IF YES: What did the doctor say?

Just before this began, were you using any medications?

IF YES: Any change in the amount you were using?

Just before this began, were you drinking or using any street drugs?

D. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, medication) or to a general medical condition (e.g., hypothyroidism)

IF THERE IS ANY INDICATION THAT THE DEPRESSION MAY BE SECONDARY (I.E., A DIRECT PHYSIOLOGICAL CONSEQUENCE OF A GMC OR SUBSTANCE, GO TO *GMC/SUBSTANCE,* A.43, AND RETURN HERE TO MAKE A RATING OF "1" OR "3."

REFER TO LIST OF GENERAL MEDICAL CONDITIONS AND SUBSTANCES, A. 4.

? 1 3
DUE TO SUB-
STANCE USE
OR GMC

A77

IF UNKNOWN: Has there been any other time when you were (depressed/OWN WORDS) like this but were not (using SUBSTANCE/ill with GMC)?

PRIMARY
MOOD
EPISODE

-> IF YES: GO TO *PAST MAJOR DEPRESSIVE EPISODE,* A. 12 AND CHECK WHETHER THERE HAS BEEN ANY OTHER MAJOR DEPRESSIVE EPISODE NOT DUE TO A SUBSTANCE OR GENERAL MEDICAL CONDITION. IF SO, ASK ABOUT THAT EPISODE.

-> IF NO: GO TO *CURRENT MANIC EPISODE,* A. 18

CON-
TINUE

?=inadequate information 1=absent or false 2=subthreshold 3=threshold or true

(Did this begin soon after someone close to you died?)

E. The symptoms are not better accounted for by [Simple] Bereavement, i.e., after the loss of a loved one, the symptoms persist for longer than 2 months or are characterized by marked functional impairment morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms or psychomotor retardation.

? 1

3

A78

SIMPLE
BEREAVE-
MENTAT
LEAST
ONE
EPI-
SODE
NOT
SIMPLE
BE-
REAVE-
MENT

IF UNKNOWN: Has there been any other time when you were (depressed/OWN WORDS) like this that did not occur after someone close to you died?

-> IF YES: GO TO *PAST MAJOR DEPRESSIVE EPISODE,* A. 12 AND CHECK WHETHER THERE HAS BEEN ANY OTHER MAJOR DEPRESSIVE EPISODE THAT WAS NOT BETTER ACCOUNTED FOR BY BEREAVEMENT. IF SO, ASK ABOUT THAT EPISODE.

-> IF NO: GO TO *CURRENT MANIC EPISODE,* A. 18.

CON-
TINUE

MAJOR DEPRESSIVE EPISODE
CRITERIA A, C, D, AND E
ARE CODED "3"

1

3

A79

GO TO
*CUR-
RENT
MANIC
EPI-
SODE,*
A. 18PAST
MAJOR
DE-
PRES-
SIVE
EPI-
SODE

How old were you when (PAST MAJOR DEPRESSIVE EPISODE) started?

Age at onset of Past Major Depressive Episode coded above

—

—

A80

How many separate times in your life have you been (depressed/OWN WORDS) nearly every day for at least two weeks and had several of the symptoms that you described, like (SXS OF WORST EPISODE)?

Total number of Major Depressive Episodes (CODE 99 IF TOO NUMEROUS OR INDISTINCT TO COUNT)

—

—

A81

NOTE: TO RECORD DETAILS OF OTHER PAST EPISODES, GO TO J. 9 (OPTIONAL).

?=inadequate information 1=absent or false 2=subthreshold 3=threshold or true

PAST MANIC EPISODE

MANIC EPISODE CRITERIA

NOTE: IF CURRENTLY ELEVATED OR IRRITABLE MOOD BUT FULL CRITERIA ARE NOT MET FOR A MANIC EPISODE, SUBSTITUTE THE PHRASE "Has there ever been another time..." IN EACH OF THE SCREENING QUESTIONS BELOW.

Have you ever had a period of time when you were feeling so good, "high," excited, or hyper that other people thought you were not your normal self or you were so hyper that you got into trouble?

(Did anyone say you were manic?) (Was that more than just feeling good?)

IF NO: What about a period of time when you were so irritable that you found yourself shouting at people or starting fights or arguments? (Did you find yourself shouting at people you really didn't know?)

A. A distinct period of abnormally and persistently elevated, expansive, or irritable mood...

Check if:
☐ elevated, expansive mood
☐ irritable mood

? 1 2 3

A126

GO TO
***DYS-
 THYMIC
 DISOR-
 DER,***
 A. 38

A127
 A128

When was that?

What was it like?

How long did that last? (as long as one week?) (Did you have to go into a hospital?)

....lasting at least one week (or any duration if hospitalization is necessary)

? 1 2 3

A129

GO TO
***PAST
 HYPOMANIC
 EPISODE***
 A. 33

Have you had more than one time like that? (Which time was the most extreme?)

IF UNCLEAR: Have you had any times like that in the past year?

NOTE: IF THERE IS EVIDENCE FOR MORE THAN ONE PAST EPISODE, SELECT THE "WORST" ONE FOR YOUR INQUIRY ABOUT PAST MANIC EPISODE. IF THERE WAS AN EPISODE IN THE PAST YEAR, ASK ABOUT THAT EPISODE EVEN IF IT WAS NOT THE WORST.

?=inadequate information 1=absent or false 2=subthreshold 3=threshold or true

SCID-I (DSM-IV) Past Manic (FEB 1996 FINAL) Mood Episodes A. 29.

<p>FOCUS ON THE WORST PERIOD OF THE EPISODE THAT YOU ARE INQUIRING ABOUT.</p> <p>IF UNCLEAR: During (EPISODE), when were you the most (OWN WORDS FOR MANIA)?</p> <p>During that time...</p>		<p>B. During the period of mood disturbance, three (or more) of the following symptoms have persisted (four if the mood is only irritable) and have been present to a significant degree:</p>				
<p>..how did you feel about yourself?</p> <p>(More self-confident than usual?) (Any special powers or abilities?)</p>	<p>(1) inflated self-esteem or grandiosity</p>	?	1	2	3	A130
<p>..did you need less sleep than usual?</p> <p>IF YES: Did you still feel rested?</p>	<p>(2) decreased need for sleep (e.g., feels rested after only three hours of sleep)</p>	?	1	2	3	A131
<p>..were you much more talkative than usual? (Did people have trouble stopping you or understanding you? Did people have trouble getting a word in edgewise?)</p>	<p>(3) more talkative than usual or pressure to keep talking</p>	?	1	2	3	A132
<p>..were your thoughts racing through your head?</p>	<p>(4) flight of ideas or subjective experience that thoughts are racing</p>	?	1	2	3	A133
<p>..were you so easily distracted by things around you that you had trouble concentrating or staying on one track?</p>	<p>(5) distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli)</p>	?	1	2	3	A134
<p>..how did you spend your time? (Work, friends, hobbies?) (Were you so active that your friends or family were concerned about you?)</p> <p>IF NO INCREASED ACTIVITY: Were you physically restless? (How bad was it?)</p>	<p>(6) increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation</p> <p>Check if:</p> <p>___ increase in activity</p> <p>___ psychomotor agitation</p>	?	1	2	3	A135
						A136
						A137

?=inadequate information 1=absent or false 2=subthreshold 3=threshold or true

During this time...

...did you do anything that could have caused trouble for you or your family? (Buying things you didn't need?) (Anything sexual that was unusual for you?) (Reckless driving?)

(7) excessive involvement in pleasurable activities which have a high potential for painful consequences (e.g., the person engages in unrestrained buying sprees, sexual indiscretions, or foolish business investments)

? 1 2 3

A138

AT LEAST THREE "B" SXs ARE CODED "3" (FOUR IF MOOD ONLY IRRITABLE)

1

3

A139

IF NOT ALREADY ASKED: Has there been any other time when you were (high/irritable/OWN WORDS) and had even more of the symptoms that I just asked you about?

-> IF YES: RETURN TO *PAST MANIC EPISODE,* A. 28, AND INQUIRE ABOUT WORST EPISODE.

-> IF NO: GO TO *DYSTHYMIC DISORDER,* A. 38.

NOTE: DSM-IV criterion C (i.e., does not meet criteria for a Mixed Episode) has been omitted from the SCID.

CONTINUE

IF NOT KNOWN: At that time, did you have serious problems at home or at work (school) because you were (SYMPTOMS) or did you have to go into a hospital?

D. The mood disturbance is sufficiently severe to cause marked impairment in occupational functioning or in usual social activities or relationships with others, or to necessitate hospitalization to prevent harm to self or others, or there are psychotic features.

1

3

A140

IF NOT ALREADY ASKED: Has there been any other time when you were (high/irritable/OWN WORDS) and had (ACKNOWLEDGED MANIC SYMPTOMS) and you got into trouble with people or were hospitalized?

DESCRIBE:

-> IF YES: RECODE CRITERION D as "3"

-> IF NO: GO TO *PAST HYPOMANIC CRITERION C,* A. 35.

CONTINUE
ON NEXT
PAGE

?=inadequate information 1=absent or false 2=subthreshold 3=threshold or true

SCID-I (DSM-IV) Version 2.0, Past Manic (FEB 1996 FINAL) 4001 Episodes A. 31

Just before this began, were you physically ill?

IF YES: What did the doctor say?

Just before this began, were you taking any medications?

IF YES: Any change in the amount you were taking?

Just before this began, were you drinking or using any street drugs?

E. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, medication) or to a general medical condition

IF THERE IS ANY INDICATION THAT THE MANIA MAY BE SECONDARY (I.E., A DIRECT PHYSIOLOGICAL CONSEQUENCE OF A GMC OR SUBSTANCE, GO TO *GMC/SUBSTANCE,* A.43, AND RETURN HERE TO MAKE A RATING OF "1" OR "3."

NOTE: MANIC-LIKE EPISODES THAT ARE CLEARLY CAUSED BY SOMATIC ANTIDEPRESSANT TREATMENT (E.G., MEDICATION, ECT, LIGHT THERAPY) SHOULD NOT COUNT TOWARD A DIAGNOSIS OF BIPOLAR I DISORDER BUT ARE CONSIDERED SUBSTANCE-INDUCED MOOD DISORDERS, A. 45.

REFER TO LIST OF GENERAL MEDICAL CONDITIONS AND SUBSTANCES, A. 21.

1 3
DUE TO SUB-
STANCE USE
OR GMC

A14

IF UNKNOWN: Has there been any other time when you were (high/irritable/OWN WORDS) and were not (using SUBSTANCE/ill with GMC)?

-> IF YES: RETURN TO *PAST MANIC EPISODE,* A. 28, AND INQUIRE ABOUT OTHER EPISODE.

-> IF NO: GO TO *DYSTHYMIC DISORDER,* A. 38.

PRIMARY
MOOD
EPISODE

CONTINUE

?=inadequate information 1=absent or false 2=subthreshold 3=threshold or true

MANIC EPISODE CRITERIA A, B, D, AND E ARE CODED "3"		1	3	A14
		GO TO *DYSTHY- MIC DIS- ORDER,* A. 38	PAST MANIC EPI- SODE	
How old were you when (PAST MANIC EPISODE) started?	Age at onset of Past Manic Episode coded above	—	—	A14
How many separate times in your life were you (HIGH/OWN WORDS) and had [ACKNOWLEDGED MANIC SYMPTOMS] for a period of time (or were hospita- lized)?	Number of Manic Episodes (CODE 99 IF TOO INDISTINCT OR NUMEROUS TO COUNT)	—	—	A14
	NOTE: TO RECORD DETAILS OF OTHER PAST EPISODES, GO TO J. 14 (OPTIONAL)	GO TO NEXT MODULE		

?=inadequate information 1=absent or false 2=subthreshold 3=threshold or true

PAST HYPOMANIC EPISODE

HYPOMANIC EPISODE CRITERIA

(When you were [HIGH/IRRITABLE/
OWN WORDS], did it last for
at least four days?)

A. A distinct period of sustained ? 1 2 3
elevated, expansive, or irritable
mood, lasting throughout at least
4 days, that is clearly different
from the usual nondepressed mood

A145

What was it like?

Check if:
— elevated, expansive mood
— irritable mood

GO TO
*DYS-
THYMIC
DISOR-
DER,*
A. 38

A146
A147

Have you had more than one time
like that? (Which time was the
most extreme?)

NOTE: IF THERE IS EVIDENCE
FOR MORE THAN ONE PAST EPISODE,
SELECT THE "WORST" ONE FOR YOUR
INQUIRY ABOUT PAST HYPOMANIC
EPISODE. IF THERE WAS AN EPISODE
IN THE PAST YEAR, ASK ABOUT THAT
EPISODE EVEN IF IT WAS NOT THE
WORST.

IF UNCLEAR: Have you had any
times like that in the past year?

FOCUS ON THE WORST PERIOD OF
THE EPISODE THAT YOU ARE
INQUIRING ABOUT.

B. During the period of mood
disturbance, three (or more)
of the following symptoms
have persisted (four if the
mood is only irritable) and
have been present to a sig-
nificant degree:

IF UNCLEAR: During (EPISODE),
when were you the most (OWN
WORDS FOR HYPOMANIA)?

During that time...

..how did you feel about
yourself?

(1) inflated self-esteem ? 1 2 3
or grandiosity

A148

(More self-confident than
usual?) (Any special powers
or abilities?)

..did you need less sleep
than usual?

(2) decreased need for ? 1 2 3
sleep (e.g., feels rested
after only three hours of
sleep)

A149

IF YES: Did you still
feel rested?

..were you much more talkative
than usual? (Did people have trou-
ble stopping you or understanding
you? Did people have trouble
getting a word in edgewise?)

(3) more talkative than ? 1 2 3
usual or pressure to keep
talking

A150

..were your thoughts racing
through your head?

(4) flight of ideas or sub- ? 1 2 3
jective experience that
thoughts are racing

A151

?=inadequate information 1=absent or false 2=subthreshold 3=threshold or true

SCID-I (DSM-IV) Version 2.0 Past Hypomanic (FEB 1996 FINAL) Mood Episodes

During this time...

..were you so easily distracted by things around you that you had trouble concentrating or staying on one track? (5) distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli) ? 1 2 3 A152

..how did you spend your time? (Work, friends, hobbies?) (Were you so active that your friends or family were concerned about you?) (6) increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation ? 1 2 3 A153

IF NO INCREASED ACTIVITY: Were you physically restless? (How bad was it?)

Check if:
☐ increase in activity
☐ psychomotor agitation

..did you do anything that could have caused trouble for you or your family? (Buying things you didn't need?) (Anything sexual that was unusual for you?) (Reckless driving?) (7) excessive involvement in pleasurable activities that have a high potential for painful consequences (e.g., (the person engages in unrestrained buying sprees, sexual indiscretions, or foolish business investments) ? 1 2 3 A154

NOTE: BECAUSE OF THE DIFFICULTY OF DISTINGUISHING NORMAL PERIODS OF GOOD MOOD FROM HYPOMANIA, REVIEW ALL ITEMS CODED "3" IN CRITERIA A AND B AND RECODE ANY EQUIVOCAL JUDGMENTS.

AT LEAST THREE "B" SXs ARE CODED "3" (FOUR IF MOOD ONLY IRRITABLE)

1 3 A155

IF NOT ALREADY ASKED: Has there been any other time when you were (high/irritable/OWN WORDS) and had even more of the symptoms that I just asked you about?

-> IF YES: RETURN TO *PAST HYPOMANIC EPISODE,* A. 33, AND INQUIRE ABOUT THAT EPISODE.

-> IF NO: GO TO *DYSTHYMIC DISORDER,* A. 38.

CONTINUE
ON NEXT
PAGE

?=inadequate information 1=absent or false 2=subthreshold 3=threshold or true

PAST HYPOMANIC CRITERION C

<p>IF NOT KNOWN: Is this very different from the way you usually are? (How were you different? At work? With friends?)</p>	<p>C. The episode is associated with an unequivocal change in functioning that is uncharacteristic of the person when not symptomatic</p>	<p>? 1 2 3</p>	<p>A156</p>
--	---	----------------	-------------

<p>IF NOT ALREADY ASKED: Has there been any other times when you were (high/irritable/OWN WORDS) in which you were really different from the way you usually are?</p>	<p>DESCRIBE:</p>
---	------------------

-> IF YES: RETURN TO *PAST HYPOMANIC EPISODE,* A. 33, AND INQUIRE ABOUT THAT EPISODE.

-> IF NO: GO TO *DYSTHYMIC DISORDER,* A. 38.

CONTI-
NUE

<p>IF NOT KNOWN: Did other people notice the change in you? (What did they say?)</p>	<p>D. The disturbance in mood and the change in functioning are observable by others</p>	<p>? 1 2 3</p>	<p>A157</p>
--	--	----------------	-------------

<p>IF NOT ALREADY ASKED: Has there been any other times when you were (high/irritable/OWN WORDS) and other people did notice the change in the way you were acting?</p>	<p>DESCRIBE:</p>
---	------------------

-> IF YES: RETURN TO *PAST HYPOMANIC EPISODE,* A. 33, AND INQUIRE ABOUT THAT EPISODE.

-> IF NO: GO TO *DYSTHYMIC DISORDER,* A. 38.

CONTI-
NUE

<p>IF NOT KNOWN: At that time, did you have serious problems at home or at work (school) because you were (SYMPTOMS) or did you have to go into a hospital?</p>	<p>E. The episode is not severe enough to cause marked impairment in social or occupational functioning, or to necessitate hospitalization, and there are no psychotic features</p>	<p>? 1 3</p>	<p>A158</p>
---	---	--------------	-------------

IF SEVERE ENOUGH TO REQUIRE HOSPITALIZATION, CONSIDERING RETURNING TO A. 28 AND RECODING AS PAST MANIC EPISODE. OTHERWISE, CONTINUE WITH A. 38 AND CODE "OTHER BIPOLAR DISORDER" ON D. 5.

?=inadequate information 1=absent or false 2=subthreshold 3=threshold or true

SCID-I (DSM-IV) Version 2.0 Test Hypomanic (FEB 1996 FINAL) Mood Episodes A. 36

Just before this began, were you physically ill?	F. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, medication) or to a general medical condition	?	1	3	A15
IF YES: What did the doctor say?		DUE TO SUBSTANCE USE OR GMC			
Just before this began, were you taking any medications?	IF THERE IS ANY INDICATION THAT THE HYPOMANIA MAY BE SECONDARY (I.E., A DIRECT PHYSIOLOGICAL CONSEQUENCE OF A GMC OR SUBSTANCE, GO TO *GMC/SUBSTANCE,* A.43, AND RETURN HERE TO MAKE A RATING OF "1" OR "3."				
IF YES: Any change in the amount you were taking?					
Just before this began, were you drinking or using any street drugs?	NOTE: HYPOMANIC-LIKE EPISODES THAT ARE CLEARLY CAUSED BY SOMATIC ANTIDEPRESSANT TREATMENT (E.G., MEDICATION, ECT, LIGHT THERAPY) SHOULD NOT COUNT TOWARD A DIAGNOSIS OF BIPOLAR II DISORDER BUT ARE CONSIDERED SUBSTANCE-INDUCED MOOD DISORDERS, A. 45. REFER TO LIST OF GENERAL MEDICAL CONDITIONS AND SUBSTANCES, A. 21.				
IF UNKNOWN: Has there been any other time when you were (high/irritable/OWN WORDS) and were not (using SUBSTANCE/ill with GMC)?		PRIMARY MOOD EPISODE			
-> IF YES: RETURN TO *PAST HYPOMANIC EPISODE,* A. 33, AND INQUIRE ABOUT OTHER EPISODE.					
-> IF NO: GO TO *DYSTHYMIC DISORDER,* A. 38.		CONTINUE			

?=inadequate information 1=absent or false 2=subthreshold 3=threshold or true

DSM-IV) Version 2.0 Past Hypomanic (FEB 1996 FINAL) Mood Episodes A. 37

HYPOMANIC EPISODE CRITERIA
A, B, C, D, E AND F ARE
CODED "3"

1 3

A160

GO TO *DYS- THYMIC DISOR- DER,* A. 38	PAST HYPO- MANIC EPI- SODE
--	--

How old were you when (PAST
HYPOMANIC EPISODE) started?

Age at onset of Past Hypomanic
Episode coded above

— —

A161

How many separate times in your
life were you (high/irritable/
OWN WORDS) and had [ACKNOWLEDGED
MANIC SYMPTOMS] for a period
of time?

Total number of Hypomanic
Episodes (CODE 99 IF TOO
INDISTINCT OR NUMEROUS
TO COUNT)

— —

A162

NOTE: TO RECORD DETAILS OF
PAST EPISODES, GO TO J. 18
(OPTIONAL)

GO TO NEXT MODULE

?=inadequate information 1=absent or false 2=subthreshold 3=threshold or true

Appendix G

Response Styles Questionnaire, Ruminative Response Scale - short form

People think and do many things when they feel down. Please read each of the items below and indicate whether you never, sometimes, often, or always think or do each of the following things when you feel down, sad, or depressed. Please indicate what you *generally* do, not what you think you should do.

1. Go someplace alone to think about your feelings

Almost never Sometimes Often Almost always

2. Isolate yourself and think about the reasons why you feel sad

Almost never Sometimes Often Almost always

3. Go away by yourself and think about why you feel this way

Almost never Sometimes Often Almost always

4. Write down what you are thinking about and analyze it

Almost never Sometimes Often Almost always

5. Listen to sad music

Almost never Sometimes Often Almost always

6. Think "Why do I always react this way?"

Almost never Sometimes Often Almost always

7. Think about a recent situation, wishing it had gone better

Almost never Sometimes Often Almost always

8. Think about how angry you are with yourself

Almost never Sometimes Often Almost always

Appendix H

PSWQ

Please indicate, by circling the appropriate number, how much each statement is typical of you.

1 2 3 4 5
(not at all typical of me) (somewhat) (very typical of me)

1. If I do not have enough time to do everything, I do not worry about it.

1 2 3 4 5

2. My worries overwhelm me.

1 2 3 4 5

3. I do not tend to worry about things.

1 2 3 4 5

4. Many situations make me worry.

1 2 3 4 5

5. I know I should not worry about things, but I just cannot help it.

1 2 3 4 5

6. When I am under pressure, I worry a lot.

1 2 3 4 5

7. I am always worrying about something.

1 2 3 4 5

8. I find it easy to dismiss worrisome thoughts.

1 2 3 4 5

Appendix I

Family History Screen

Please tell me the first names and ages of your biological mother and father. Now tell me the names, ages, and sexes of all the children born to (*mother's first name*) and (*father's first name*). Please start with the first born (*oldest*) and include yourself in the list. (*Fill out information on separate sheet. For each question, a response is registered for each family member.*)

Has anyone on the list ever had a serious mental illness, emotional problem, or nervous breakdown?
Who was that? Anyone else? (*until no more names are given*)

Has anyone on the list ever seen a psychiatrist, psychologist, social work, doctor, or other health professional for a psychological or emotional problem?
Who was that? Anyone else? (*until no more names are given*)

Has anyone on the list ever stayed overnight or longer in a hospital or treatment facility because of any mental or emotional problem?
Who was that? Anyone else? (*until no more names are given*)

Has a doctor ever given anyone on the list any medicine for a psychological or emotional problem?
Who was that? Anyone else? (*until no more names are given*)

Has anyone on the list ever had difficulty carrying out their usual responsibilities, such as working, going to school, or taking care of the family or household?
Who was that? Anyone else? (*until no more names are given*)
Other than physical illness, was anyone unable to carry out their usual responsibilities for a week or more?
Who was that? Anyone else? (*until no more names are given*)

Did anyone on the list ever feel sad, blue or depressed for most of the time for two days or more?
Who was that? Anyone else? (*until no more names are given*)
Without including times of physical illness or mourning after a death, did anyone have a period during which they felt sad, blue or depressed that lasted two weeks or more?
Who was that? Anyone else? (*until no more names are given*)

Did anyone on the list ever have a period of feeling quite tired, having less energy, or not caring about their usual activities?
Who was that? Anyone else? (*until no more names are given*)
Don't include times of physical illness, or mourning after a death. Other than that, did anyone feel very tired most of the time, have no energy, or not care about their usual activities, for at least two weeks?
Who was that? Anyone else? (*until no more names are given*)

Did anyone on the list ever have sleep problems, like trouble falling asleep, or waking up too early, or sleeping too much?
Who was that? Anyone else? (*until no more names are given*)
Was it as much as an hour a night for two weeks or more, and not because of a physical illness?
Who was that? Anyone else? (*until no more names are given*)

Has anyone on the list ever had a period of feeling extremely happy or high?

Who was that? Anyone else? (*until no more names are given*)

I mean “high as a kite”, so that other people worried about them, or so that it interfered with carrying out normal responsibilities. Has anyone been unusually happy or high, not because of drugs or alcohol, for two days or more?

Who was that? Anyone else? (*until no more names are given*)

Has anyone on the list ever has a period in which they were more active or talkative than normal?

Who was that? Anyone else? (*until no more names are given*)

I mean extremely over-talkative, so that people worried about them, or so that it interfered with carrying out normal responsibilities. Has anyone been like that, without being under the influence of drugs or alcohol, at least two days?

Who was that? Anyone else? (*until no more names are given*)

Has anyone on the list ever tried to kill him or herself, or made a suicide attempt?

Who was that? Anyone else? (*until no more names are given*)

This may be a painful question, but did the person actually kill him or herself?

Who was that? Anyone else? (*until no more names are given*)

Appendix J

Word Stimulus List

NEGATIVE

cemetery
coffin
debt
depressed
anguished
fever
gloom
grief
jail
lonely
massacre
mosquito
neglect
poverty
robber
starving
stupid
toothache
unhappy
useless

NEUTRAL

chin
errand
habit
humble
banner
jelly
kerchief
lamb
pamphlet
barrel
reserved
statue
basket
thermometer
umbrella
violin
bathroom
bland
blase
cabinet

POSITIVE

angel
caress
cute
delight
diamond
easygoing
grateful
hug
impressed
luxury
melody
rainbow
reward
sailboat
scholar
sweetheart
twilight
untroubled
warmth
wit